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DISEASES OF CHICKENS

J. R. BEACH and M. A. STEWART

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CONTENTS

DISEASES OF CHICKENS[1, 2]

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GENERAL CONSIDERATIONS OF DISEASE AND OF CONTROL MEASURES

TYPES OF POULTRY DISEASE

DISEASES OF POULTRY may be grouped into three general types: (1) transmissible diseases caused by specific infectious agents known as bacteria, viruses, or fungi (molds) and animal parasites, and certain diseases which appear to be transmissible but for which a specific cause has not been found; (2) diseases due to specific deficiencies of certain essential ingredients in the diet; and (3) a group termed "nonspecific," which includes those diseases and abnormal conditions not as yet shown to be associated with infection, parasitism, or dietary deficiencies.

Infectious and parasitic diseases have been the most serious causes of poultry mortality, and it is these which have received the most attention since the seriousness of poultry diseases was recognized and intensive study of them began, about thirty years ago. Knowledge since accumulated, however, has made available to poultry raisers control measures by which the losses from the majority of these diseases may be kept within reasonable limits. Diseases of this classification and the means for their prevention and control constitute the subject with which this bulletin is principally concerned.

The diseases definitely related to nutrition are principally those resulting from vitamin deficiencies. The existence of such diseases on poultry farms in epidemic proportion was first recognized about 1920 and they have become of increasing significance as methods of rearing and feeding poultry have become more and more artificial. Knowledge of poultry nutrition, however, has kept pace with dietary deficiency diseases, and most of this type of trouble can be avoided. In fact, some

[1] Received for publication March 11, 1942.

[2] This bulletin supersedes Agricultural Extension Circular 8, *Diseases and Parasites of Poultry in California*, by J. R. Beach and S. B. Freeborn. That circular, after five editions, is now out of print. The material included in the present bulletin is strictly factual as far as possible. In cases in which knowledge still is incomplete, an attempt has been made to present differences of opinion in unbiased fashion. Most emphasis is given to the disease problems of greatest economic importance to commercial poultry raisers of California. A major portion of the material presented is based upon work done at the California Agricultural Experiment Station, but information obtained from the work of others has been freely used and is hereby acknowledged. The inclusion of extensive bibliographic references, however, has been omitted.

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of the nutritional diseases had been seen in the laboratory before they were known to have occurred on farms.

There is a tendency to ascribe to the feed much of the sickness among poultry which may appear suddenly or which is of an obscure nature, but in most cases it is impossible to determine that the feed is at fault. Sickness caused by a spoiled ingredient in a ready-mixed mash has been encountered occasionally, however, and losses from food poisoning in

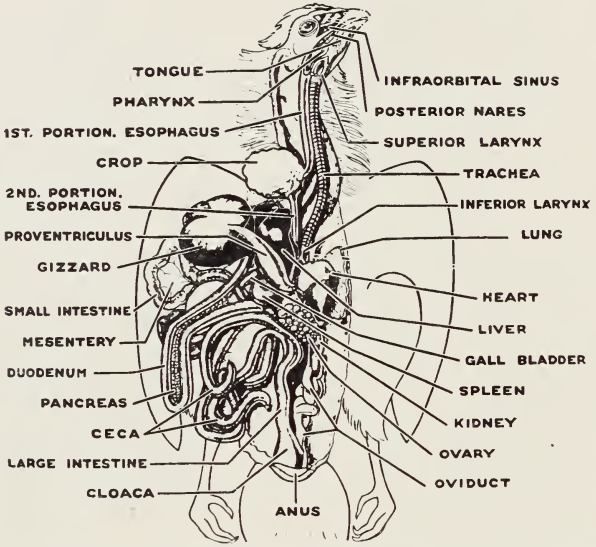

Fig. 1.—Diagram showing the location of anatomical parts of chickens.

flocks which are fed kitchen refuse or garbage are fairly common. In the latter case the poisonous material is usually spoiled canned fruit, vegetables, or meat, which causes a disease known as "botulism" or "limberneck."

Rations which are compounded from ingredients of good quality on the basis of present knowledge of poultry nutrition are not likely to be the cause of sickness and death. But rations designed to stimulate very high egg production or very rapid growth are apt to be responsible for loss of birds. Poultrymen should be guided by the recommendations of poultry departments of the agricultural experiment stations,[5] or other reliable sources of information, to ensure that their feeding practices are sound, and that they secure the benefit of the new developments in poultry nutrition.

[5] For the recommendations of the poultry department of this station, see: Almquist, H. J., T. H. Jukes, and W. E. Newlon. Feeding chickens. California Agr. Ext. Cir. 108:1–41. Revised 1940.

BREEDING FOR DISEASE RESISTANCE

The possible application of selective breeding to the reduction of poultry disease and mortality is well stated in the following excerpts from Bulletin 626:

"Some poultrymen are prone to suggest that, if birds are properly selected and mated, strains will be created with such inherent vigor that they can withstand adverse conditions commonly found on poultry ranches. But this seems more than can logically be expected to result from good breeding. Inadequate or deficient rations, insanitary conditions, and generally poor management cannot be overcome by better breeding of poultry."

"Some experimental evidence of the inheritance of resistance to specific infections in chickens has been obtained. Roberts and Card (1926 and 1936) have demonstrated inherited differences in susceptibility to pullorum disease, and Lambert and Knox (1932) obtained similar results with fowl typhoid. They failed, however, to obtain strains in which all the birds were resistant. Furthermore, such diseases can be more efficiently controlled by other means. On the other hand, there is evidence on the hereditary nature of resistance to certain other disease conditions for which methods of prevention and control are not yet known. This applies particularly to neurolymphomatosis (paralysis) and may also be true of other forms of lymphomatosis (big-liver disease or leukemia), leucosis, and certain types of tumors." [See p. 78.]

"Losses from pathology associated with the digestive system in the 1933 flock were unusually high and were found predominantly in the families from one male and his son. After elimination of these families, losses from disorders of the alimentary system dropped sharply for the succeeding years."

"All available evidence indicates that disease resistance is specific in nature. Creating a strain resistant to one disease does not mean that the strain will be resistant to any other disease. Nor does it mean that the strain will be unusually susceptible to some other disease. Genetically, every disease presents a separate problem of breeding for resistance. Whether or not genetics can be effectively applied to the control of all diseases is questionable."

"Breeding for disease resistance is slow and costly. Where adequate means are available to control diseases by means of agglutination tests, vaccines, specific drugs, or sanitation, trying to breed for resistance is uneconomic. Where no means of control are available and where there are evidences of different responses by birds of different strains or families, genetic selection is advisable, even though improvement of the stock comes slowly."[6]

That mortality from nonspecific disease or functional disturbances of vital organs may be influenced by breeding for longevity is indicated by additional excerpts from the same source.

[6] Taylor, L. W., and I. M. Lerner. Breeding for egg production. California Agr. Exp. Sta. Bul. 626:21, 22, 23, 43, 44. 1938.

"Just how much . . . . mortality is due to inherited factors cannot be estimated accurately, but there is good evidence in many cases that at least part of the mortality up to the completion of the first laying year is controlled by genetic factors, and hence the condition is amenable to improvement by proper breeding methods."

". . . . Greater vitality will ensue in a flock as a consequence of selection of longer-lived strains. Differences in mortality between families in the same flock are known to persist for several generations, as they have in the California Station flock, and hence are probably heritable. Despite the poultryman's interest in obtaining long-lived birds, comparatively few reports are available on the results of selection for longevity. This fact may indicate that breeding for characters influencing longevity is a complex process."

TYPES OF MEASURES FOR THE CONTROL OF INFECTIOUS DISEASES AND PARASITES

There are two types of measures or procedures which are useful in the prevention and control of infections and parasitic diseases. The first consists of the specific measures, such as vaccination for fowl pox or spraying with an oil to destroy mites, which are effective against a specific germ or parasite. These are taken up later in the discussions of individual diseases. The second type includes the more general measures which are embraced by the term "sanitation" and which apply to all.

In its broader aspect, sanitation may be defined as providing a place in which chickens may live safely and produce efficiently. More specifically, it consists of protecting them from undue exposure to infection and parasites. The effective application of sanitary measures must, therefore, be based upon knowledge of how infection and parasitism are spread from fowl to fowl or from pen to pen on a farm, perpetuated on a farm from year to year, and introduced on a farm for the first time.

Different diseases are spread in very different ways. Transmission of infection from one bird to another or from pen to pen may be by birds actively diseased or by birds that have nearly or entirely recovered from some disease but which continue to carry the disease-producing agent. These latter are often referred to as "latent cases of infection" or as "healthy carriers." This is the common means by which respiratory diseases are spread and perpetuated on a farm.

Certain diseases are known to be spread and transmitted by insect vectors, such as flies and mosquitoes. In districts where the latter are very plentiful, fowl pox, which is spread by them, is more difficult to control.

The sanitary procedure for a farm must be designed to cope with the particular disease problem which is present. For example, the separation of young birds from the old may be of greater importance in controlling

a certain disease or preventing its spread than maintaining proper cleanliness. In many cases, however, it is extremely difficult or impossible to determine how the disease is introduced or is carried from one place to another. Therefore, it is necessary to employ general sanitary measures which apply to any type of infection.

Cleaning and disinfection, if properly done, are usually, but not always, adequate to make a building in which an infectious disease has occurred safe for further use. However, there are some disease-producing agents, such as coccidia and the virus of fowl pox, which are so resistant that they would not be destroyed by disinfection as ordinarily practiced. In this case, the mechanical removal of infection by cleaning is of really more importance than the application of disinfectants. That is why, in a cleaning and disinfection process, the cleaning should be given as much emphasis as disinfection. The two are complementary; but the thorough mechanical cleaning is, perhaps, the more important, at least when dealing with resistant organisms.

SANITARY CONSIDERATIONS IN THE CONSTRUCTION, EQUIPMENT, AND CARE OF POULTRY HOUSES

The type of poultry house is not so important as that it possess those features of design and arrangement that are essential to sanitation. Floors and foundations should be impervious to moisture, have a *smooth,* hard surface to facilitate cleaning, and be rat- and vermin-proof; concrete is the most suitable material. Wood floors are more difficult to clean and they furnish shelter for rats and vermin. Dirt floors are objectionable because they cannot be adequately cleaned or disinfected. The walls should be constructed of matched lumber so as to provide a smooth surface and be free from cracks that would permit draughts and in which mites and ticks could find lodging places. The openings to admit light and for ventilation should be covered with wire netting to exclude wild birds; or, better still, with wire screen to exclude insects as well as birds. They should be large enough to admit plenty of light, fresh air, and also direct sunlight, exposure to which is beneficial to fowls and destructive to bacteria. Facilities for ventilation that will ensure an abundance of fresh air day and night in all kinds of weather should be provided. The extent to which especial attention must be given to ventilation depends upon the type of the building, the size of the pens, and the number of fowls kept in a pen.[7]

Droppings boards are optional. If they are installed, chickens should

[7] For recommendations on poultry housing, see: Dougherty, J. E., and H. L. Belton. Poultry houses and equipment. California Agr. Exp. Sta. Bul. 476:1–77. Revised, 1940, by H. L. Belton and V. S. Asmundson.

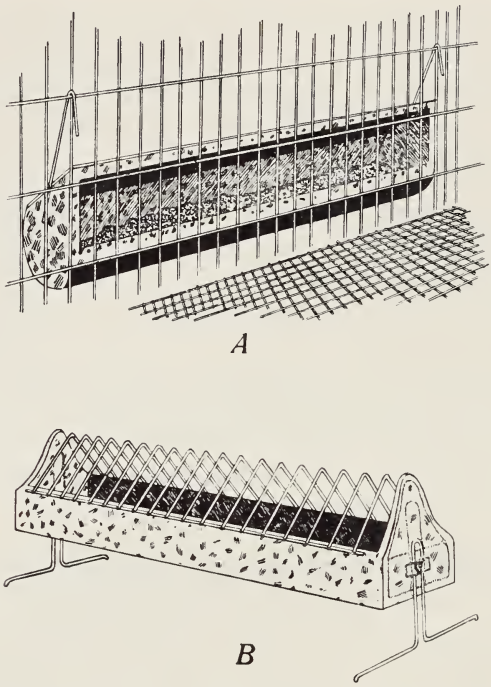

Fig. 2.—*A*, A sanitary type of metal feed hopper designed for hanging on the outside of a wire-enclosed sun porch; *B*, a type of metal feed hopper with wire guard to exclude chickens and prevent soiling of feed with droppings. (From Bul. 613.)

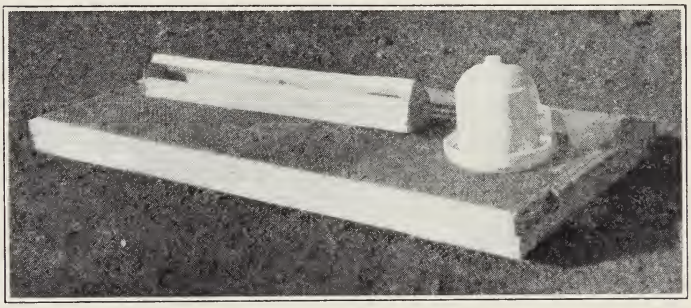

Fig. 3.—Feed hopper and waterer suitable for chicks. The wire platform aids in keeping the adjacent area in a sanitary condition. (From Bul. 613.)

be excluded from them by wire netting beneath the roosts. If they are not installed, litter will have to be changed more often, which is likely to be more costly than the daily cleaning of droppings boards would be. Furthermore, the fertilizer value of droppings undiluted with litter will be lost and all eggs laid by chickens while on the roost will fall to the

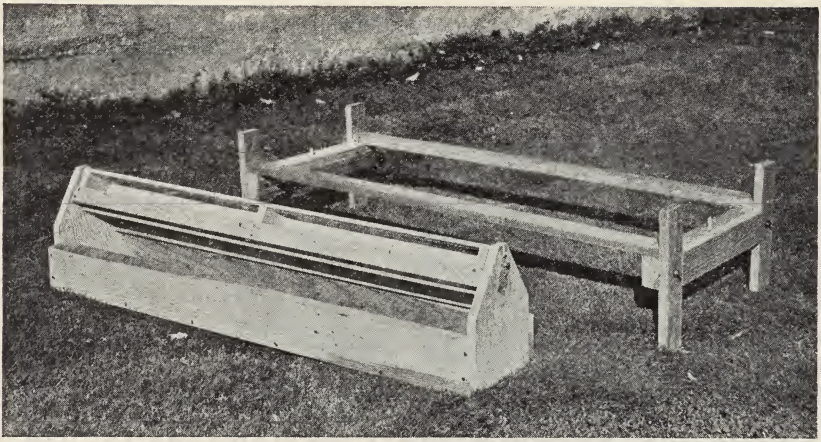

Fig. 4.—A sanitary homemade type of feed hopper of wood construction with a revolving crosspiece to prevent chickens from roosting above the feed; adaptable for use inside a house or on range. (From Bul. 476.)

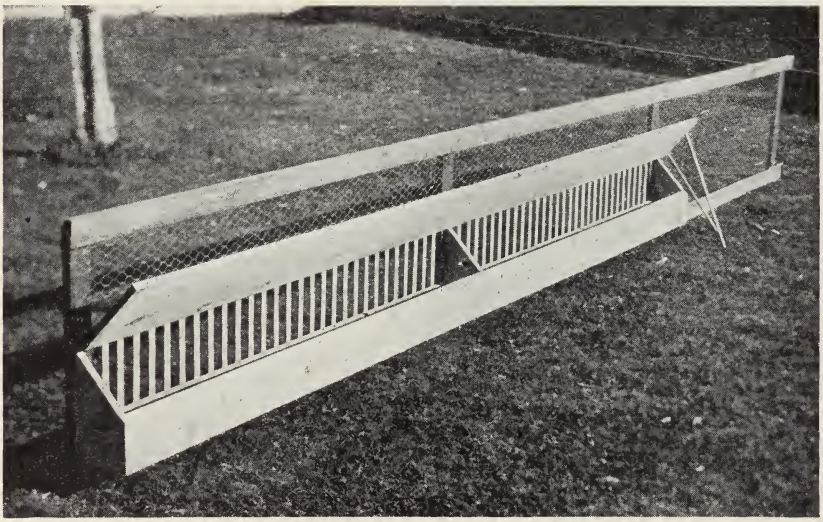

Fig. 5.—A sanitary homemade feed hopper that can be installed on the outside of a wire fence or sun porch or in the front wall of a poultry house. Feed is replenished from the outside. (From Ext. Cir. 58.)

floor and be eaten by the other chickens. The latter may cause some of the chickens to become egg eaters and may be the means of spreading infections, such as pullorum disease (p. 26), which are transmitted through eggs.

Feed troughs or hoppers should be of a type that will prevent fowls from getting in them or roosting over them, and that can be easily cleaned (figs. 2–5). Removable hoppers are preferable to built-in ones.

Devices for drinking should be designed to supply fresh, clean water continuously, and to prevent the floors around from becoming dampened by overflow or by water scattered by fowls (fig. 6). A continuous

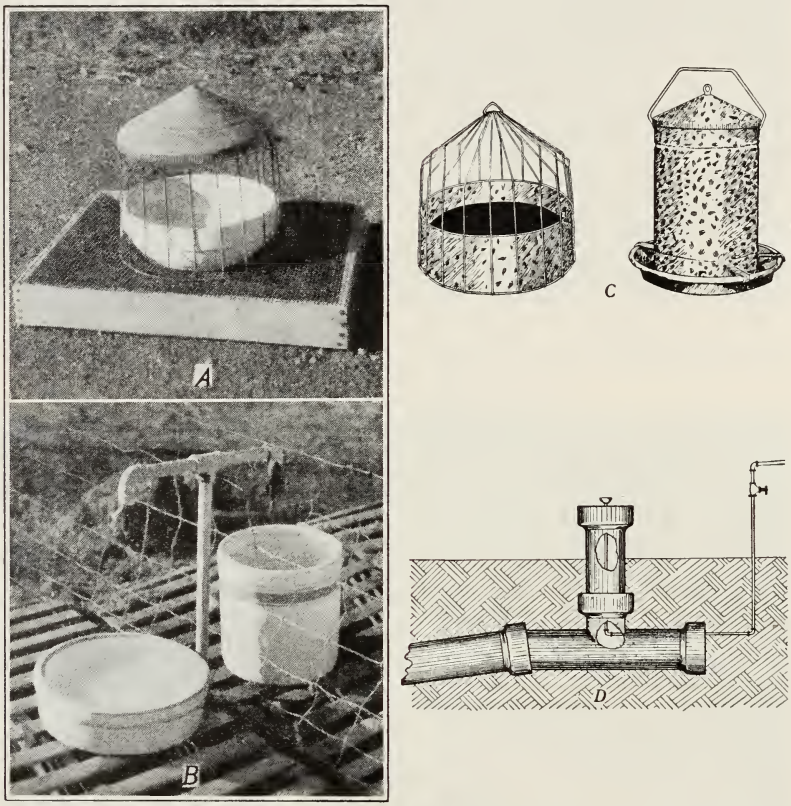

Fig. 6.—*A*, A sanitary type of water container placed on a wire-mesh platform. This can easily be made automatic by piping water to it and controlling the flow by means of a float valve. *B*, A method of supplying chickens with running water; the overflow passes into a drainage pit below the lath platform. It could be made more sanitary by putting a wire guard around the vessel as in *A* and by covering the platform with wire mesh. *C*, Two types of sanitary waterers made of galvanized metal. The one on the left could easily be made automatic. *D*, A good method of supplying water to chickens. The overflow from the cup is carried through the drain tile to an outside pit or sewer. A desirable improvement would be increasing the length of the vertical water pipe and drain tile and surrounding it with a wire-mesh-covered platform. (From Bul. 613; original for *D* by courtesy of L. Van Es, University of Nebraska.)

flow or drip system, arranged so that pollution by droppings cannot occur, is preferable to open troughs or pans. Water containers should be surrounded by a wire-mesh-covered platform to exclude fowls from litter or areas of floor which have become moistened with water scattered by

the birds when drinking. The exclusion of chickens from the moist areas around watering devices, in either the houses or the yards, is an important factor in general sanitation, and is absolutely essential in the prevention of intestinal parasitism.

When the contour of the land permits, building sites should be selected that will provide free natural drainage away from the houses. If this is not possible, a drainage system should be installed.

A regular schedule for cleaning operations should be arranged and adhered to as carefully as for any other farm operation. Droppings should be removed from droppings boards daily, whether or not fowls are excluded from them by wire netting, and stored in flyproof bins or maggot traps (p. 130) or hauled a considerable distance from the houses.

Litter, in the form of the cheapest absorbent material available, such as straw, shavings, or rice hulls, should be liberally supplied. If at all feasible, it should be renewed once a week through the first laying year, that is, until the birds are about 18 months old. This is the period of greatest danger from infections and parasitic diseases. If there is no special infection problem to be dealt with, the litter for older birds ordinarily will not require renewal oftener than once a month unless, in the meantime, it becomes damp. Litter changes should be made more often in houses which have no droppings boards, when fowls are confined to the houses, and when there is a large number of birds per unit of floor space.

A house should be regularly cleaned and disinfected in the thorough manner described later, not less than twice each year and at such other times as it may become empty. Trash, such as loose boards and empty feed sacks, should not be allowed to accumulate in or around poultry houses. The cleaning of premises in preparation for disinfection, either as a routine procedure or after an outbreak of an infectious disease, is described in the section on disinfection.

SANITATION OF POULTRY YARDS

Soil upon which chickens are kept always constitutes the most difficult problem in poultry sanitation. Many types of disease germs discharged with the excretions of infected birds find in soil a suitable medium for their preservation until they may be taken up again by other chickens. Soil, once infected, may remain so for varying lengths of time, according to the nature of the infection, the character of the soil, and the conditions of weather or climate. Left unpopulated, the infection in the soil will gradually die out, but for some types of infection the time required for this to occur is greater than any normal period of disuse of a yard on a poultry farm. Therefore, even the practice of leaving brooder yards idle

from one brooding season to the next or the rotation in use of yards is likely to be inadequate for purification of contaminated soil. Disinfection of soil by means of chemicals is, as a rule, of uncertain value. A better treatment, and one which may remove enough of the contamination to make it worth the effort, is to sweep up and haul away the surface dirt and accumulated droppings, particularly from the portion of the yard adjacent to the houses.

Certain diseases, such as coccidiosis and intestinal worm infestation, are so widespread that few poultry farms have escaped their ravages. As time goes on, soil contamination is likely to build up, make control of those diseases more difficult, and losses greater.

Such experiences have persuaded poultrymen to try raising their chickens without direct contact with soil as a means of protecting them from soil-borne diseases. The procedure has proved to be of much benefit and also satisfactory from a husbandry standpoint. Both field experience and experimentation have clearly demonstrated that chickens do not obtain any nutrient from soil or crops growing in the yards which cannot be supplied in the feed, and that exposure to sunshine is the chief if not the only benefit which chickens derive from being out of doors. Consequently, small yards, surfaced with concrete or asphalt, or sun porches, with the tops, bottoms, and sides made of wire netting, have replaced soil yards on a large number of farms. From a sanitation aspect, the sun porches are preferable. Usage has shown that the hard-surfaced yards are difficult to keep clean unless covered with litter, and in warm weather they become so hot that the chickens do not use them.

These substitutes for soil yards will not entirely eliminate soil-borne diseases and parasites, and unless proper attention is given to their cleanliness and sanitation, their benefit may be lost. They will, however, reduce the likelihood of extensive outbreaks of such diseases, make it easier to control any that do occur, and progressively reduce the problem. Therefore, raising chickens without direct contact with soil from birth through the first laying year and even after, if desired, is highly recommended.

DISINFECTION AND DISINFECTANTS[8]

Disinfection means the act of destroying the cause of infection and is based on our knowledge of the existence of disease germs. These germs are extremely small and may remain for an indefinite time in dust, cracks, and crevices, and in small particles of manure or other material

[8] For additional information on disinfection, see:

Pope, George W. The disinfection of stables. U. S. Dept. Agr. Farmers' Bul. 954:1–12. 1932.

Bonnikson, H. P. Disinfection of dairy stables. California State Dept. Agr. Mo. Bul. 19(5):352–55. 1930.

that may stick to the floor, to the walls of buildings, or to feed troughs or other accessories. Efforts to remove and destroy them must, therefore, be exceedingly thorough to be effective.

Direct Sunlight.—Sunlight is a natural agency that will destroy or weaken the infective properties of some viruses and bacteria and the eggs of many parasitic worms. Thus the value of placing the openings in poultry houses to permit the entrance of as much sunlight as possible is evident. Sunlight, however, cannot be regarded as more than an accessory to other means of destroying disease germs.

Heat.—Heat is one of the most effective and most used disinfectants available, but the possibilities for its application to poultry-house disinfection are limited. Some of the ways in which it may be utilized are as follows:

Very hot water is destructive to bacteria and, where available direct from a pipe line, can be utilized both for cleaning and disinfection. When it is practicable to equip poultry houses with a hot-water line from a steam boiler, the problem of cleaning and disinfection is greatly simplified.

A jet of live steam can be utilized for disinfection when available. Steam is ineffective a few inches away from the nozzle, however, and the nozzle must be held close to the object against which it is directed and moved slowly and systematically about until the entire surface has been touched and heated to a boiling temperature. Merely filling a room with steam is valueless.

Heat can also be applied by means of a "fire gun" (a large blowtorch) to concrete floors and, to a more limited extent, to other parts of buildings and metal accessories. Its use should be restricted to types of buildings in which it would not create a fire hazard, to concrete yards, metal feed hoppers, metal water vessels, and similar pieces of equipment. Although the flame itself is intensely hot, a comparatively long exposure must be given to raise the temperature of an object or surface to be disinfected high enough to destroy all bacteria present. *To be effective, the flame should be moved very slowly and systematically over the entire surface of the floor or equipment being treated.* Stafseth of the Michigan Agricultural Experiment Station has estimated that the disinfection of concrete floors with this apparatus would require 1 hour for each 180 square feet (approximately) of floor area. The fire gun can be used advantageously as an adjunct to chemical disinfection for burning up particles of litter or droppings not removed by the cleaning process. Its use on soil is of doubtful value because it cannot be relied upon to raise the temperature of the soil sufficiently to such a depth as to make the effort worth while.

A steam sterilizer of the type used for milking equipment on dairy farms can also be adapted to poultry farms for sterilizing accessories and equipment, such as feed hoppers, drinking vessels, feeding buckets, and nests. Gas or electric heating units can be obtained separately and a sterilizer cabinet can be built from galvanized metal to any size and shape desired. This is perhaps the most practical and effective means of utilizing heat for disinfection on poultry farms.

Disinfecting with Chemicals.[9]—Certain chemicals which have the power to destroy disease germs must be depended upon for the most part in the practical work of disinfection. The selection of suitable and dependable disinfectants is no easy matter. They vary greatly in character and in their fitness for the purpose in view. An ideal disinfectant would be: (1) highly effective against a large variety of germs; (2) highly soluble in hard as well as soft water; (3) retentive of germicidal action after the exposure to air and contact with organic matter that occurs when it is applied; (4) relatively nonpoisonous to man and farm animals; (5) nondestructive of metals and fabrics; (6) of such composition that it does not leave an objectionable residue or odor; (7) reasonable in cost. Not all of these properties will be found in any one chemical disinfectant. Therefore, in selecting one for a particular purpose, the objectionable features as well as the germ-destroying power of the preparation must be given consideration. For example, a disinfectant which leaves an oily residue might be satisfactory for a poultry house but not suitable for an incubator or incubator room.

Disinfectants of similar composition are sold under a variety of trade names. Before purchasing an unfamiliar brand, one should carefully examine the label and make comparisons with some well-known product of the same type. The "phenol coefficient" usually given on the label is a fair, although not entirely satisfactory, means of comparison. To protect the buyer against inferior products, federal and state laws require that the labels of disinfectants shall contain no statement, design, or device which is false, fraudulent, or misleading in any particular. Samples of commercial disinfectants are collected and tested, and when evidence of adulteration or mislabeling is found, proper legal action is instituted. These laws have less control, however, over statements

[9] Some of the materials mentioned in this bulletin may become scarce or unavailable for civilian use because of the war. Where two or more chemicals are effective in the prevention, control, or treatment of a disease, all have been mentioned. If the reader finds that none of these are available now, he will naturally do the best he can with any substitutes he can obtain. Great caution should, however, be exercised in the use of substitutes, particularly those untested for the proposed service. When in doubt about the advisability of using certain materials as substitutes for those recommended, readers should consult the farm advisors in their respective counties or the Agricultural Extension Service, University of California, Berkeley, California.

made by salesmen, by radio, or other advertising matter. Buyers, therefore, should accept only those statements of the worth of disinfectants that are given on the label of the original container. Good guides in the selection of a disinfectant for general farm use are lists of the products tested and approved by the United States Bureau of Animal Industry and by the Division of Animal Industry of the California State Department of Agriculture for use in official disinfection.[10] The label on the container of a product which has been given such official approval will usually give a statement to that effect. A disinfectant should be diluted according to the directions given on the label.

Details of Disinfection.—In order for a chemical disinfectant to kill germs, it must come in close contact with them. If germs are protected by a film of grease or albuminous matter or embedded in bits of dry manure or dirt, the contact of the disinfectant with them is prevented and the germs escape destruction. Furthermore, germs are very tiny and a small bit of the discharges from diseased fowls or of other material can harbor millions of them. This means that surfaces to be disinfected must be mechanically cleaned before action from the disinfectant can be expected.

All the nests, feed troughs, hoppers, and other accessories should be emptied and brushed out. All of the surfaces, such as ceilings, walls, partitions, floors, and droppings boards, should be swept until free from cobwebs and dust. All litter, manure, and refuse from houses and yards should be removed to a considerable distance and, in the case of a cleanup after a disease outbreak, be burned if possible. After sweeping and scraping as thoroughly as possible, the dried pieces of manure or dirt that are still stuck to the floor or walls should be scrubbed off. For this purpose a hot lye solution (see p. 18) is recommended. The building is now ready for disinfection.

The best method of applying disinfectant is with a strong spray pump. A high-pressure power-operated orchard spraying outfit is recommended. It is well to have the pump equipped with not less than 15 feet of hose, to which is attached a 5-foot length of iron pipe with a spray nozzle at the end. With such equipment the operator can proceed rapidly and reach all parts and surfaces easily. The disinfectant will have greater effectiveness if applied hot. It is advisable for the operator to wear rubber boots, heavy rubber or leather gloves, a wide-brimmed felt or rubber hat, long-sleeved work clothing or a rubber coat. Equipped

[10] The disinfectants recognized for official use are listed in: Permitted disinfectants. U. S. Bur. Anim. Indus. Cir. Letter 2010. This may be obtained by writing to the U. S. Bureau of Animal Industry, Washington, D. C.

A list of the disinfectants offered for sale within the state, with their analyses, can be obtained from the Division of Chemistry, State Department of Agriculture, Sacramento, California.

in this manner, he can do a thorough job without any great danger of the disinfectant's coming in contact with his skin.

The time element is an important factor in the effectiveness of disinfectants. For example, a disinfectant in the concentration used may kill bacteria in 10 minutes but not in 5 minutes, or may kill certain types of bacteria in 5 minutes and others in not less than 10 minutes. To ensure an effective job, therefore, all surfaces and crevices must be so heavily saturated with the disinfectant that they will remain wet for a considerable time. Care must be taken that the more inaccessible places, such as the bottom surfaces of droppings boards and the interior and bottoms of nests, feed hoppers, and other attached fixtures, are not neglected.

The characteristics and uses of some of the common disinfectants are given on the following pages.

Phenol, or Carbolic Acid.—Phenol, a well-known coal-tar derivative with a characteristic odor familiar to everyone, is a solid in the pure state and, therefore, is usually sold in water solutions. It is a good disinfectant in 2 per cent to 5 per cent solution but is too expensive for general use. It is rather caustic and must be carefully handled. "Phenol coefficients" of other disinfectants are obtained by a standard procedure of comparing their germicidal activity with that of phenol.

Cresol, or Cresylic Acid.—Cresol is a yellow or brown liquid derivative of coal-tar, with high disinfectant activity. Since it is only slightly soluble in water, it is seldom used in the pure state, but is the active principle of many of the best brands of commercial disinfectants.

Crude Carbolic Acid.—Crude carbolic acid should not be confused with pure carbolic acid (phenol) or with cresol. It consists of a mixture of phenol, cresol, and other coal-tar derivatives which are inert. Since it is of uncertain composition and of low solubility in water, it is not a satisfactory disinfectant. It may be used as a substitute for oil in spraying a poultry house for the control of mites and ticks.

Saponified Cresol Solutions.—Water-soluble preparations consisting of mixtures of cresol or cresylic acid and soap are called saponified cresol solutions. They are very stable and efficient preparations and are used extensively for official disinfection. The most refined preparation of this type, Saponated Solution of Cresol U.S.P. is rather expensive and, therefore, the cheaper substitutes are used for general farm disinfection. The United States Bureau of Animal Industry specifies that saponified cresol solutions applied under its supervision must contain not less than 50 nor more than 53 per cent cresol and not less than 21 per cent soap; must form clear solutions in water and be used in the proportion of 4 fluid ounces per gallon of water. Poultrymen should purchase only saponified cresol solutions that meet these specifications.

Sheep-Dips.—The coal-tar disinfectants known as sheep-dips form milky solutions in water and vary greatly in their solubility and disinfecting value. For most purposes on poultry farms, they are less desirable than saponified cresol solutions.

Chlorine Disinfectants.—Chlorine disinfectants are powdered or solutions of hypochlorites prepared with chlorine gas. They are sold under a great many trade names. Their value is dependent upon their content of available chlorine, which should be at least 2.6 per cent by weight. These preparations, although highly destructive to bacteria, are not regarded as desirable for general disinfection because chlorine becomes inactive in the presence of organic matter. Chlorine disinfectants are very useful, however, for the disinfection of water supplies, or of water troughs or fountains, feed hoppers, or other equipment with which the use of a preparation of cresol might be objectionable. They should be used according to directions on the container.

Chlorine gas has not been found of value for disinfection of poultry quarters by fumigation or in the treatment of flocks for a respiratory disease.

Iodine.—Iodine, a highly effective disinfectant, is adapted for use on poultry houses and equipment only in a colloidal form called "iodine suspensoid." This has been reported by the Michigan Agricultural Experiment Station to be very destructive not only to bacteria but also to coccidia and worm eggs which are strongly resistant to other disinfectants. For this reason, iodine suspensoid is recommended by the manufacturer[11] particularly for disinfecting brooder-house floors and equipment. Like chlorine, its action is quickly lost in the presence of organic matter and therefore it should be applied only to surfaces after they have been thoroughly washed.

Chlorinated Lime (Chloride of Lime).—Chlorinated lime, prepared by saturating slaked lime with chlorine gas, is used principally as a bleaching powder or as a deodorant. It should contain from 30 to 35 per cent of available chlorine. The United States Bureau of Animal Industry recognizes chlorinated lime containing at least 30 per cent available chlorine for official disinfection when used in proportions of 1 pound to 3 gallons of water. Products containing less available chlorine should be used in more concentrated solutions. Fresh solutions must be prepared daily. All products containing chlorine must be handled with care, for chlorine is destructive to fabrics, leather, and metal.

Quicklime (Unslaked Lime, Calcium Oxide).—A fairly good germicide, quicklime may be applied with a brush or spray as whitewash. It is not recommended except to lighten the interior of dark poultry houses

[11] Merck & Co., Inc.

or as a covering for rough lumber. Its disinfectant action may be increased by the addition of chlorinated lime or saponified cresol solution. Since quicklime is very caustic, it must be handled carefully and poultry kept away until whitewashed surfaces are thoroughly dry.

Slaked lime has no disinfectant properties.

Lye (Sodium Hydroxide).—A 2 per cent solution of lye is an excellent disinfectant; in fact, it is more active against some disease germs than saponified cresol solutions. It is also an excellent cleaning agent. The use of a 2 per cent solution in hot water (1 pound to 6¼ gallons or one 13-ounce can to 5 gallons) is recommended for cleaning and disinfecting poultry houses. Since it is very caustic, users should wear rubber boots and loose-fitting rubber gloves, be careful not to get any on their clothing or skin, and have with them some vinegar for immediate application to any portion of the skin with which the lye solution may come in contact.

Copper Sulfate (Bluestone).—Although copper sulfate and some other copper salts are very destructive to algae and some fungi, they are much less destructive to bacteria, and, consequently, they are not considered as good general disinfectants. The usefulness of copper sulfate, therefore, is limited to treatment of drinking water and for cleaning water vessels and areas around them in combatting fungus diseases. Drinking water containing more copper sulfate than 1 part in 500 may be toxic to birds and also may make the water so distasteful that consumption is seriously reduced.

Potassium Permanganate.—Potassium permanganate, although having no usefulness as a general disinfectant and no medicinal value, is convenient and safe for use as an antiseptic in drinking water, is inexpensive, and relatively nonpoisonous to poultry. One level teaspoon in each gallon of drinking water will aid in preventing the spread of infection by the water. When the solution loses its germicidal property, its color changes from purple to brown, and it should then be renewed.

Bichloride of Mercury (Corrosive Sublimate).—Bichloride of mercury is a powerful disinfectant in a 1 to 1,000 solution in water. It is usually dispensed in tablet form and with directions stating the amount of water in which one tablet should be dissolved to make a solution having the desired strength. It has the disadvantages of being a violent poison, of corroding metals, and of uniting with albuminous substances to form inert compounds. Unlike the disinfectants previously described, corrosive sublimate leaves no odor and on this account is useful where odors would be undesirable. It must be handled with great caution to avoid accidental poisoning of stock, dogs, or cats, and the solutions must be prepared in wooden or earthenware containers. Water or feed

troughs to which it has been applied should be washed with fresh water before they are used again.

Sodium Orthophenylphenate.—This substance has only recently been recommended as a general disinfectant. It has no objectionable odor, is relatively nontoxic, is highly efficient for most disease germs, and is readily soluble in water. It may be purchased in the form of a grayish, brownish, or white powder or flakes, which must be kept in a closed container to prevent deterioration. It is now sold under several trade names, which are included under the United States Bureau of Animal Industry list of permitted disinfectants. It gives best results when applied hot.

Formalin and Formaldehyde Gas.—Formaldehyde gas is an excellent disinfectant available through usual trade channels as formalin, a watery solution containing not less than 37 per cent of the gas. The latter has high germicidal properties in 5 to 10 per cent solution, but is objectionable for use as a general disinfectant because the fumes which it gives off are very irritating to the eyes and nose. It is relatively nonpoisonous to animals, does not harm metals or fabrics, and leaves no residue or lasting odor. It is useful, therefore, in the disinfection of water vessels, feed hoppers, and other poultry-house accessories.

Formalin finds its principal use as a source of formaldehyde gas for disinfection by fumigation of any building that can be made nearly airtight. Few, if any, poultry houses, however, can meet this requirement. Fumigation in a small, tightly closed room would be practicable for clothing and other equipment that are difficult to disinfect by other methods. The usual means for liberating formaldehyde gas is by the chemical reaction that occurs when formalin is mixed with potassium permanganate. The procedure is as follows:

For each 1,000 cubic feet of air space, 16⅔ ounces of potassium permanganate crystals or powder is placed in a wide pan; 20 ounces of formalin is then poured upon it and the room immediately closed for 12 hours or longer. Since considerable heat is generated by the chemical reaction, it is well to place the pan containing the chemicals in a larger pan containing water. The temperature in the room should not be less than 65° F and the humidity not less than 60 per cent.

At present, formaldehyde gas is used rather extensively for the disinfection of incubators of the forced-draft type. Fumigation is not so well adapted to the compartment, or still-air, type of incubator except between hatches because of the slowness with which the gas would escape from a compartment. Fumigation of forced-draft incubators is said to be safe between hatches, while eggs are in the incubator, and even when chickens are hatching out. This is neither a safe nor a successful practice unless a correct procedure is exactly followed. It is best to

follow instructions obtained from the manufacturer of the incubator to be disinfected.

The directions for two methods of incubator fumigation based on Ohio Agricultural Extension Bulletin 90[12] and Illinois Agricultural Experiment Station Circular 403,[13] are as follows:

Potassium permanganate method: 1. For each 100 cubic feet of incubator space, use 70 cc of formalin and 35 grams of potassium permanganate if the fumigation is done between hatches or on the seventeenth or eighteenth day of incubation. Use only one half of the above amounts of chemicals if the incubator is fumigated when chicks are hatching.

2. Place the potassium permanganate in an enamelware pan (never in a glass or pottery container) that holds at least ten times the amount of chemicals used, and put this in the control compartment of the incubator below the fan. More than one pan may be necessary for large incubators.

3. Pour the formalin over the potassium permanganate and close the incubator door at once. Leave the door closed for 3 hours. No change in the ventilation of the incubator is necessary.

The cheesecloth method: 1. Measure out 40 cc of formalin for each 100 cubic feet of incubator space. Use only one half of this amount if the incubator is fumigated while chicks are hatching.

2. Cut a piece of cheesecloth large enough to absorb all of the formalin (1½ yards for each 100 cc).

3. Completely absorb the formalin with the cheesecloth and hang it under the fan in the incubator. For large incubators, hang a strip of cheesecloth under each fan. Close the door and start the fan. Do not open the door for 3 hours. No change in the ventilation of the incubator is necessary.

Fumigation of incubators while chicks are hatching is a somewhat hazardous procedure. If it is done, the cubic content of the incubator should be very accurately computed and the correct amount of formalin carefully measured out. Never fumigate chicks more than 48 hours old.

With the potassium permanganate method, make one fumigation when 5 to 10 per cent of the chicks are out of the shell and two more, 12 hours apart. Three or 4 hours after each fumigation, remove all chicks that are dry.

With the cheesecloth method, fumigate twice, once when from 10 to 20 per cent of the eggs are hatched and again 12 to 15 hours later. Three or 4 hours after each fumigation, remove all dry chicks.

[12] Dakan, E. L., and Fred Speer. Sanitation in the hatchery. Ohio Agr. Ext. Bul. 90:1–16. 1929.

[13] Graham, Robert, and V. M. Michael. Incubator hygiene in the control of pullorum disease. Illinois Agr. Exp. Sta. Cir. 403:1–16. 1933.

During fumigation by either method, the incubator temperature should be 90° to 100° F and the wet-bulb thermometer reading, 90° to 95°.

Paraffin-Oil Emulsion.—Paraffin-oil emulsion is a very effective spray for killing ticks, mites, and certain other external parasites which hide in the cracks and crevices of poultry houses during at least part of their lives. It is prepared by first dissolving 1 pound of soap in 10 quarts of hot water. After the soap has dissolved, the solution is removed from the fire and allowed to cool to a lukewarm temperature. Then 10 quarts of paraffin oil are added to the soap solution and thoroughly stirred until the liquid becomes creamy. This constitutes the stock solution. The spray is made by diluting 1 part of the stock solution with 6⅓ parts of water.

Lime-Sulfur Dip.—Lime-sulfur dip is employed in the control of certain external parasites, as described in the section of this publication dealing with parasitic diseases. It may be made up at home by using 8 pounds of quicklime and 24 pounds of flowers of sulfur to 100 gallons of water. The lime is slaked with sufficient water to form a thick paste and the sulfur is then sifted in and mixed thoroughly with a hoe. This mixture is placed in a kettle with 30 gallons of water and boiled for 2 hours. Prepared "lime-sulfur" dips can be purchased on the market and should be used according to the directions given on the container.

Kerosene Emulsion.—Kerosene-emulsion oil spray is sometimes used, as described later in the section dealing with external parasites, to control certain species of poultry pests. It is made by first dissolving ½ pound of soap in 1 gallon of hot water; if the water to be used is hard, a little borax should be added to soften it. After the soap is dissolved, the solution is taken from the fire and allowed to cool. Then 2 gallons of kerosene are added and, with a small hand spray pump having a fine nozzle, the mixture is drawn into the pump and out through the nozzle back into the container from which it was drawn until it becomes so thick as to go hard through the pump. This stock solution is diluted with 5 parts of water to make the finished spray. The stock solution, if well prepared, should keep for at least a month.

Wettable Sulfur.—Finely ground sulfur to which has been added a material which will increase its suspensibility in water is called "wettable sulfur." Commercial preparations are readily available.

Creolin.—Creolin is a mixture of high-boiling coal-tar oils and phenols rendered emulsifiable with water by a special process. Creolin (veterinary) is employed in the control of certain external parasites of poultry.

Anthracene Oil.—Anthracene oil, a coal-tar oil that is heavier than creosote, sometimes known as "carbolineum," is effective in controlling ticks and mites in poultry houses.

MANAGEMENT PRACTICES IN PREVENTING AND CONTROLLING TRANSMISSIBLE DISEASES

The discussion of prevention and control of disease thus far has been concerned principally with the elimination of the spread of infection through contaminated material in houses and equipment by cleanliness and disinfection. Consideration will now be given to practices of a different nature which are necessary for the elimination of some of the other and equally dangerous means by which infection may be introduced to or spread about a farm. Failure to put these practices into operation may nullify the benefit from a carefully conducted program of cleaning and disinfection.

Infection may be spread by anything, living or inanimate, which may pass, by design or chance, from one poultry farm to another or between different pens of chickens on the same farm. Some of these, such as wild birds and dust carried by the wind, are wholly or partially uncontrollable, but this is not true of chickens added to a flock, poultry crates, visitors, poultry buyers, and many others. Some of the precautionary measures that may be adopted to prevent the spread of disease by such agencies are given in the following paragraphs.

Measures to Prevent Introduction of Infection to a Farm.—1. For the establishment of new flocks or in making additions to existing ones, hatching eggs or day-old chicks are not likely to introduce any infection except pullorum disease or similar infections which may be transmitted through eggs, and this can usually be avoided by a careful investigation of the source of the chicks (see p. 26–38).

2. Partly grown or mature chickens, including breeding males or females, should not be used to establish a new flock or be added to an existing flock unless one can be certain that they have not been exposed to or affected with some infection which they may still be carrying. It is possible for such chickens, although apparently healthy, to carry infective material mechanically on feathers, feet, or other parts of the body; to be in the incubation stage of a disease to which they have recently been exposed during transit or while exhibited at shows or fairs, or to be healthy carriers of disease, that is, to still carry the causative agent of a disease, such as infectious coryza, infectious laryngotracheitis, or fowl cholera, with which they had been infected and apparently recovered. The last of these has been the more common means by which disease has been brought to a farm by seemingly healthy chickens. Purchase of chickens under a guarantee of freedom from a transmissible disease is not necessarily good insurance because the seller may not have a clear understanding of the disease condition in his flock. For example,

he may have a mild form of infectious coryza (cold) in his flock and regard this as a harmless type of cold or may have had an outbreak of infectious laryngotracheitis in the flock and not know that recovered chickens may carry the virus of this disease indefinitely.

3. If partly grown or mature chickens are purchased, they should be kept separated from other chickens for at least 2 weeks to allow for the development of any acute disease with which they may recently have been infected. The same applies to chickens returned from a show, fair, or contest. This precaution, however, is not adequate to reveal the presence of healthy carriers among the new chickens.

When a small group of new breeding birds is brought onto a poultry farm, the safest procedure is to keep the new birds permanently isolated and add their progeny, instead of the new birds themselves, to the flock.

4. Breeding chickens should be purchased subject to a negative reaction to the test for pullorum disease unless they come from a flock which, by having been systematically tested over a period of years, has been shown to be free from this disease. Chickens from a pullorum-disease-free flock which have been entered in a show or contest should be tested for pullorum disease before they are returned to the flock.

5. Visitors, particularly those whose business or occupation takes them from farm to farm, should not be permitted to enter poultry houses or yards.

6. Poultry buyers' crates should not be allowed on a poultry farm unless they have been thoroughly cleaned and disinfected since last used, and even then had best be kept outside of the houses and yards. The poultry buyers should likewise be excluded from the houses. Crates belonging on the farm should be cleaned and disinfected each time they are used for taking chickens to market.

7. All openings in the walls of the houses, except doors and chicken exits, should be covered with wire netting of a mesh small enough to exclude wild birds. The possibility of infection being introduced by wild birds can be further reduced by the use of wire mesh-covered sun porches instead of open yards.

8. Areas of a farm on which poultry houses are located should be well fenced to prevent chickens from escaping to neighboring farms, from which they might bring back disease, and also to keep out chickens which may stray from neighboring flocks.

9. Measures to keep down flies and mosquitoes should be adopted. This may require not simply the proper disposal of manure and the elimination of breeding places of mosquitoes by the individual poultryman but also community action and the establishment of mosquito-abatement districts.

Measures to Prevent the Spread and Recurrence of Disease on a Farm.—The recommendations which follow are of particular importance as aids in preventing the recurrence of certain diseases among the growing stock and laying pullets on a farm each year. Some of these are perpetuated by contaminated houses, yards, and equipment; others by cases of latent infection or healthy carriers among the older chickens which have been infected previously.

1. All houses and equipment should be thoroughly cleaned and disinfected before they are used for young chickens.

2. Land should not be used for a pullet range if it has been recently occupied by adult chickens or is close to pens of adult chickens.

3. Pullets not raised on range should be confined to houses and wire-mesh sun porches. The litter in the houses should be renewed at least once a week until the end of the first laying year.

4. Pullets should be housed entirely separate from older birds. Empty sections of a house partially filled with adults should not be used for pullets.

5. On large farms, separate attendants and equipment for pullets and for adult birds should be provided.

6. If indications are seen of a disease against which sanitation is particularly effective, precautionary measures should be increased by such means as more frequent renewal of litter, more thorough and frequent cleaning and disinfection of water and feed containers, and careful inspection of the flock for the purpose of detection and removal of sick birds.

7. Any chickens that become sick should be immediately taken out of the flock and either isolated for further observation or killed and burned. By the prompt isolation of the first cases of an infectious disease, a serious outbreak may be headed off. If several cases of an unknown disease occur, specimens should be taken or sent to a veterinarian or to the nearest of the laboratories of the State Department of Agriculture or of the University for examination.[14] *Do not rely upon diagnoses made*

[14] The State Department of Agriculture laboratories are located as follows: State Office Building, Sacramento; 627 F Street, Petaluma; and 1451 Mirasol Street, Los Angeles. The University of California laboratories are in the Division of Veterinary Science at Berkeley and Davis.

In shipping specimens the sender should: (1) Be certain that the specimens selected are representative of the major illness in the flock. (2) Send sick rather than dead chickens if a selection of representative specimens can be made on the basis of symptoms. (3) Select dead chickens as soon after death as possible, thoroughly chill them in a refrigerator or by other means and wrap them in several layers of paper. (4) Ship by prepaid express. (5) Take the specimens to the express office just prior to the departure of a train that will ensure delivery to the person making the examination on the same day as shipped or in the morning after an overnight journey. (6) Attach to the package in a sealed stamped envelope a statement telling

by remedy or feed salesmen, or servicemen, or other persons untrained in the diagnosis of poultry diseases!

8. If an outbreak of an infectious disease occurs in a flock, the still healthy chickens should be moved to clean pens. New cases that develop should be immediately taken out. The detection of new cases will be facilitated by dividing the flock into as many small groups as possible.

All sick chickens should be killed and the carcasses burned; the houses, feed and water containers, and other equipment, should be thoroughly cleaned and disinfected. Yards should be cleaned and plowed and left idle for a month or more. The safety of the houses and yards for re-use may be determined by keeping a few young test chickens in them for a month before they are fully restocked.

Medication of Drinking Water.—Nonpoisonous chemical disinfectants may be added to the drinking water for flocks when an infectious disease occurs. The only benefit of this practice results from the ability of the disinfectant to destroy disease germs in the water and thus prevent disease from spreading by this medium. Contrary to common belief, such chemicals do not act as intestinal disinfectants nor otherwise exert a beneficial action after consumption by the fowls.

Chemicals for disinfection commonly used in the drinking water are potassium permanganate or one containing chlorine. The amount of the former to add is that sufficient to give a claret color to the water. More should be added when the color fades. The amount of a chlorine compound to add is determined by its content of available chlorine (as given on the label). An amount sufficient to provide 1 part of available chlorine in 3,000 parts of water may be used with safety. Dilutions of chlorine as high as 1 part in 100,000 will destroy germs but are not suitable for use in the poultry house because of the rapidity with which chlorine is inactivated in the presence of organic matter.

Copper sulfate in the drinking water has a limited usefulness as an aid in the control of certain fungus diseases (see p. 98).

Medication of the Feed.—Some nicotine preparations and possibly certain other chemicals that are used to treat chickens for intestinal worms may be adminstered mixed with the mash. Otherwise, no sound evidence of benefit from medicated mashes has been shown.

The combination of drugs known as "tonics" for mixing with the poultry feed have little, if any, value in preventing or curing infectious diseases. Possibly they may serve to stimulate the appetite of convalesc-

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the age of the chickens affected, the size of the flock, the number already affected, symptoms shown by the sick, whether the same or similar condition has previously existed on the farm and other pertinent information. (7) Give the name and address of both owner and sender, if shipment is not made by the owner.

ing flocks and thereby hasten return of the fowls to normal condition. A formula that may be used is as follows:

Pulverized gentian	1 pound
Pulverized ginger	¼ pound
Pulverized saltpeter	¼ pound
Pulverized iron sulfate	½ pound
Pulverized nux vomica	¼ pound

Add 1 ounce of the preparation to each 5 pounds of mash.

INFECTIOUS DISEASES

The diseases discussed in this section are limited to those that are of particular economic importance to the commercial poultry industry of California. Nearly all of those that occur sporadically and cause only small losses, or that, although prevalent in some parts of the world, do not occur in California, are omitted.

PULLORUM DISEASE (BACILLARY WHITE DIARRHEA)

Pullorum disease is caused by infection with a species of bacteria called *Salmonella pullorum* and affects both chicks and adult fowls. Chicks have an acute form of the disease from which many succumb after a brief illness. In adults the infection is usually localized in the ovaries and rarely produces visible symptoms.

The term "white diarrhea" is a misnomer. Diarrhea is not a characteristic or constant symptom of the disease in chicks and the frequently occurring white chalky discharge from the vent of adults is seldom related to pullorum infection.

Symptoms and Diagnosis of the Disease in Chicks.—Diseased chicks do not exhibit any symptoms that are of particular diagnostic value. A small amount of chalklike material may be seen adhering to the fluff below the vent of many of the chicks, and occasionally such collections around the vent attain considerable size. But, as stated previously, diarrhea is very often absent and is perhaps just as frequently seen accompanying conditions other than pullorum disease. A high mortality in a flock of correctly brooded chicks during the first 2 weeks of their lives strongly suggests pullorum disease but is not a definite indication of it, since there are other causes of excessive mortality among young chicks. According to some observations, an abrupt rise in the number of deaths 6 to 8 days after hatching, followed by a considerably decreased yet abnormally high mortality up to 2 weeks of age, is more suggestive of pullorum disease than a high mortality during the first few days after hatching. Contrary to common belief, pullorum disease in chicks does not always cause heavy mortality. Therefore, the disease

may be present without exciting suspicion on the part of the caretaker and thereby escape detection. An abnormally high mortality can be expected if the brooding conditions for the infected flock are poor, the diet is faulty, or the quality of the chicks is low.

The finding in dead chicks of yellow or ocher-colored livers and of unabsorbed yolks was formerly considered indicative of pullorum disease. Such findings, however, are commonly seen in chicks that die during the first week and are absent from those which die later, regardless of the cause of death. Furthermore, unabsorbed yolk is present in many normal chicks under a week old.

More characteristic changes are found in chicks which die during the second week. These consist of small gray spots in the liver, gray nodules in the heart wall, and gray or yellowish nodules in the lungs. But even these lesions are not sufficiently typical or constant in their occurrence for certain identification of the disease. Consequently, a positive diagnosis of pullorum disease can be made only by bacteriological procedures in a laboratory. These require at least 48 hours for completion.

Symptoms and Diagnosis of the Disease in Adults.—Pullorum disease is present in many flocks of adult chickens, but only occasionally does it cause an acute fatal type of disease. Probably some of the earlier reported outbreaks of this nature were in reality due to some other but closely related species of organisms, which, because of inadequate bacteriological procedures, were not differentiated from *Salmonella pullorum.*

Salmonella pullorum has been reported as causing an inflammation of the oviduct of pullets, which was manifested by eggs with an offensive odor, blood-spotted and blood-smeared eggs, soft-shelled eggs, a profuse white discharge from the vent, and the practice of cannibalism. Occurrences of this nature from pullorum infection, however, are comparatively rare. The writers have examined numerous chickens with inflamed oviducts and chalky discharge from the vent without being able to incriminate any organism as the causative factor.

Pullorum disease of adults usually becomes localized in some organ where it remains dormant and produces no external evidence of its presence. In males, it has been found most frequently in the heart sac but has been isolated also from the testes, spleen, gall bladder, and heart muscle. In females, the ovary is the organ most frequently involved. The prominent changes here are abnormal yolks (fig. 7). These abnormalities include bloody and cheesy ovules; cysts attached to the ovary; small, partially solidified blood-tinged yolks; yolks of various sizes with capsules but partially filled with thick yellow or greenish-yellow liquid; solidified, angular, yellow, greenish-yellow, or blood-tinged yolks; and

yolks with a thick opaque capsule containing yellow, semisolid, oily material or a clear yellow, oily liquid with white flakes in suspension. Caseous egg material in the oviduct is also a rather common location of the organism.

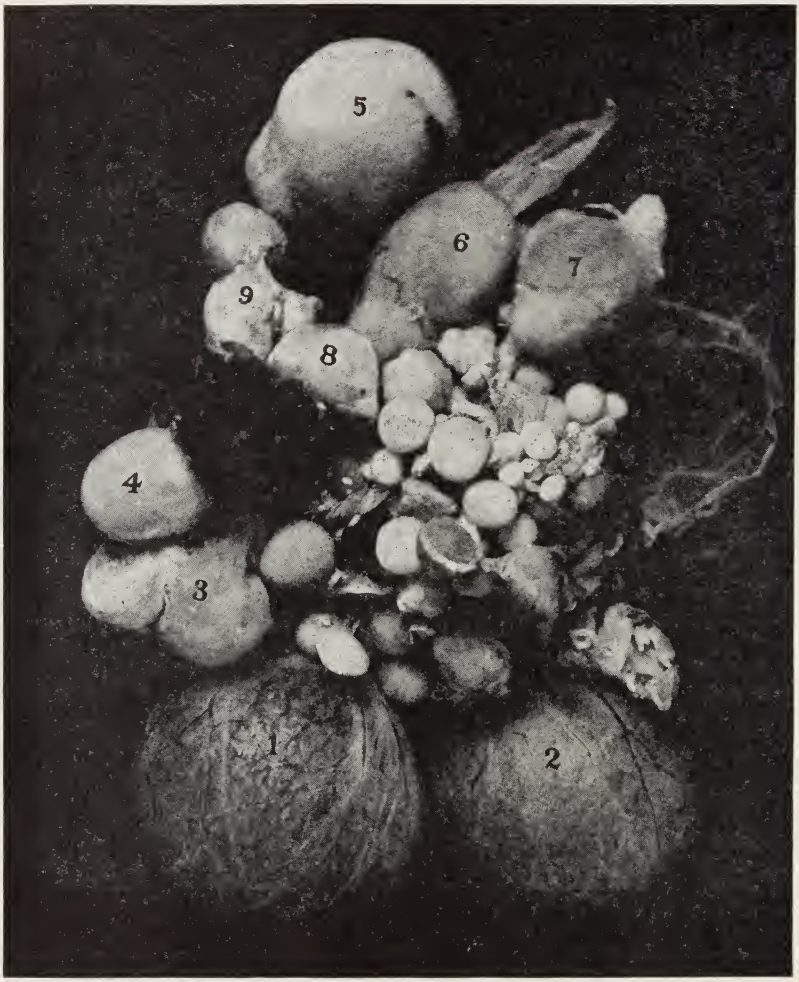

Fig. 7.—Ovaries of a hen infected with pullorum disease: nos. 1 and 2 are normal yolks; nos. 3 to 9 and many of the smaller yolks are abnormal because of the infection. Such abnormalities may also result from other causes. (From Ext. Cir. 8.)

Localization of the infection may also occur in other and sometimes very obscure places, particularly the heart sac, either in addition to or in the absence of ovarian lesions. One case has been reported in which it was isolated from the middle ear only, with no evidence of infection

in any other part. As with chicks, the finding of characteristic lesions on autopsy by no means definitely indicates the presence of infection, and *bacteriological examination must be resorted to in establishing a diagnosis.* A means of detecting pullorum disease in the living fowl is afforded by the agglutination test, discussed later.

The Effect of the Disease on Production and Reproduction.—Pullorum disease of adults, although usually localized and not responsible for the death of many birds, causes serious loss by its effect on egg production and reproduction. It has been established that infected fowls, as a rule, lay fewer eggs than noninfected fowls of like quality. The most serious consequence, however, is that living germs may be contained in hatching eggs laid by an infected hen and thus be introduced into a hatchery. When an infected fertile egg is incubated, the embryo becomes infected and either dies in the shell or survives to emerge as an infected chick, from which the infection spreads to other chicks in the incubator, chick boxes, and brooder.

Infected chicks hatched from infected eggs are the source of the infection responsible for most outbreaks among flocks of chicks. A single infected chick among a batch at the time of hatching is sufficient to infect many others before the chicks leave the incubator or after they are placed in shipping boxes or in the brooder. In forced-draft incubators the infection may be widely disseminated by means of down from the infected chicks. With the still-air type of incubator, consisting of a series of small compartments, the spread of infection within the incubator is ordinarily confined to the compartment in which the infected chicks are located. Incubator-acquired infection may enter the bodies of the chicks by way of either the digestive or the respiratory tracts. Infection by the latter route is probably responsible for the occurrence of lesions in the lungs.

Other sources of infection in chicks are contaminated incubators, shipping boxes, brooder houses, or brooding equipment, all of which can readily be eliminated by the sanitary measures discussed on pages 7–25.

Variation in the Extent of the Infection among Progeny of Infected Flocks.—Much variation has been observed in the occurrence of pullorum disease among different hatches of chicks from flocks in which there were presumably infected hens and even among different lots of chicks of the same hatch that were shipped to different poultrymen. Many breeders and hatcherymen and some investigators have, therefore, questioned whether the infected hen was really dangerous as a source of infection among chicks. Consideration of some of the following four facts regarding the infection in hens and its manner of dissemination affords explanation for variations of this nature.

1. A wide variation has been shown to exist in the frequency of the occurrence of infection in eggs laid by individual infected hens.

2. Many infected eggs fail to hatch because of infertility or death of the embryo in the shell.

3. Many of the chicks that hatch from infected eggs die or are destroyed soon after they emerge from the shell and before there has been much opportunity for dissemination of infection among the healthy.

4. In the case of difference in the amount of the disease among several lots of chicks from one large hatch, but a few of the chicks may have been infected and all of these may have been contained in one or two lots.

It is, therefore, conceivable that even though a flock may contain numerous infected hens, very few infected eggs may be produced, or may hatch, and hence loss from pullorum disease may occur infrequently or irregularly among the flock's offspring. It must be borne in mind, however, that exactly the reverse may occur. Numerous infected eggs may be laid from which infected chicks may be hatched, and conditions may be such that infection will be rapidly and widely disseminated among the others of the hatch.

Sources of the Infection in Adults.—The majority of infected adults are those which were infected as chicks and in which the bacteria became localized in some organ as previously described. Fowls can, however, acquire the infection and become carriers at any age. The practice of feeding uncooked infertile and dead-germ eggs from the incubators has produced extensive infection in some flocks. It has also been demonstrated that healthy hens may acquire infection from association with infected hens.

To what extent males may be responsible for the spread of infection among hens is not clearly understood. The actual transmission of the disease to hens by infected males has not been conclusively demonstrated. Nevertheless, an infected male must be regarded as a potential source of infection of hens with which he may be associated. There is evidence that the infection will spread more rapidly among hens when males are present in the flock than when only females are present. The role of the male in this case is probably that of a mechanical carrier of infection from hen to hen.

Control of Outbreaks of the Disease in Chicks.—There is little evidence to indicate that efforts to reduce losses from outbreaks of pullorum disease among chicks have, as a rule, been highly successful. Many of the seemingly good results from methods that have been tried are probably due to the fact that the mortality in outbreaks varies between wide limits and many times is low, and that the deaths usually have greatly decreased or have entirely ceased by the time the chicks are

from 2 to 3 weeks old. The employment of sanitary measures are undoubtedly of benefit if properly carried out. These would consist of cleaning and disinfecting houses and equipment, promptly removing and destroying sick or dead birds, and dividing the chicks into as small units as possible. This last-mentioned measure might be highly effective in preventing heavy losses if it were done early in the outbreak, if not many infected birds were left in the flock after the visibly sick ones had been removed, and if these were contained in one or two of the small units.

In controlled experiments with artificially infected chicks, drugs and chemicals, including potassium permanganate, hydrochloric acid, mercuric chloride, sulfocarbolates, sulfuric acid, resorcin, and several hypochlorite (chlorine) solutions, were shown to be of no benefit when taken into the alimentary tract and, therefore, ineffective in reducing mortality among chicks that were actually infected. Some chemicals, however, particularly the hypochlorite solutions, may be of some value as disinfectants in drinking water to prevent the spread of infection through this route. They should be used as directed on the container.

Prevention of the Disease.—As in other infectious diseases, successful prevention lies in removing the sources of infection. These are, as previously stated, contaminated incubators, shipping boxes, and brooders; also infected chicks, particularly before removal from the incubator, and infected hens.

The incubators and brooders can readily be eliminated as sources of infection by thorough cleaning and disinfection before each time they are used, and the boxes by using only new ones or by disinfection after each use.

In forced-draft incubators, as previously stated, infection may be widely disseminated at hatching time on the down from diseased chicks. This can be reasonably well overcome by fumigation with formaldehyde gas (see p. 20) at suitable intervals during the incubation period and while hatching is taking place. It is said that this procedure, if correctly done, will not decrease the hatchability of the eggs or harm the chicks and will prevent dissemination of the disease through the incubator by destroying the infection on the down of chicks that have hatched from infected eggs. This treatment, of course, does not destroy the organisms within the bodies of the infected chicks and, therefore, they remain sources of infection after they are taken from the incubator. Careful inspection of the chicks for the purpose of detecting and removing those that are weak will serve to remove many of the infected ones and will accordingly reduce the likelihood of serious losses from pullorum disease in the brooder.

The elimination of infected breeding fowls, the most serious source of pullorum disease in chicks, is a more difficult procedure because such fowls, from outward appearance, cannot be distinguished from healthy ones. The only practical means for the detection of pullorum disease carriers that has thus far been discovered is a blood test, known as the "agglutination test." This has been popularly termed the "B.W.D. test" and has been used extensively for several years. Three methods of mak-

Fig. 8.—Method of drawing a blood sample for the agglutination test. (From Ext. Cir. 8.)

ing the agglutination test have been developed: the slow serum, or tube, test; the rapid, or plate, serum test; and the rapid whole-blood test. A skin test, termed the "pullorin test" has also been tried. This test is much easier to apply than the agglutination test, but the results obtained with it have been unsatisfactory.

The Slow Serum Agglutination Test.—For the slow serum, or tube, agglutination test, from 1½ to 3 cc of blood are obtained from each fowl by puncturing the wing vein and collecting the blood in small clean vials or test tubes (fig. 8). A number, corresponding to that on the leg band of the fowl, is written on the cork or a gummed label on the vial, which is then placed in a slanting position until the blood has clotted. The vials are then placed in a cool place, preferably in a refrigerator, until the serum, or clear, straw-colored liquid portion of the blood, has separated from the clot. In case the serum does not separate

within a few hours, the clot is stirred with a toothpick and allowed to stand a few hours longer. A minute quantity, usually from 0.01 to 0.04 cc, of the serum of each blood sample is mixed with a small amount (usually 1 cc) of test fluid or antigen in a test tube. The antigen consists of 0.85 per cent salt solution containing a small amount of phenol or other preservative, to which has been added sufficient killed *Salmonella pullorum* bacteria to make the liquid slightly cloudy. The serum-antigen

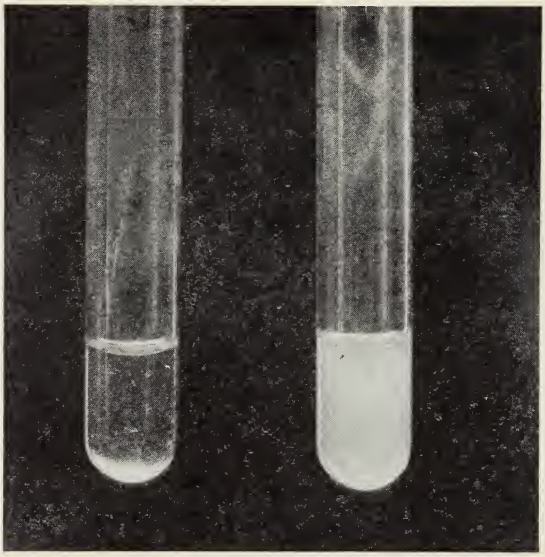

Fig. 9.—A positive (left) and negative (right) reaction to the tube, or slow, agglutination test. (From Ext. Cir. 8.)

mixture is allowed to stand for 24 hours or longer at incubator or room temperature before a reading of the results of the test is made. If the blood serum contains what is known as agglutinins, the organisms in the antigen clump together and sink to the bottom of the tube with consequent clearing of the fluid. This is known as a positive reaction (fig. 9). Most fowls with pullorum disease have agglutinins in their blood and give a positive reaction to the agglutination test.

The Rapid Serum Agglutination Test.—The rapid serum agglutination test is made on a clean glass plate which is ruled into squares of about 1 inch with a diamond-pointed or wax pencil. Blood serum is obtained in the same manner as for the slow agglutination test. A small amount (0.02 to 0.04 cc) of blood serum is transferred to a square on the glass plate where it is mixed with a drop (0.04 to 0.05 cc) of antigen. This antigen has a concentration of bacteria about fifty times that of

the antigen for the slow agglutination test. The glass plate is placed over a black background or used as a cover for a box, the interior of which can be illuminated. The reaction, that is, clumping of the bacteria, is completed and can be read in from 1 to 15 minutes (fig. 10).

The results of comparisons of the rapid test with the slow test indicate that the two are equally satisfactory in the detection of carriers of *Salmonella pullorum*. The advantage of the rapid one is that extensive laboratory facilities are not required and the test results are obtained more quickly. Considerable experience is required, however, to interpret the reactions correctly. Veterinarians or others undertaking this work

Fig. 10.—A positive (left) and negative (right) reaction to the rapid serum agglutination test. (From Ext. Cir. 8.)

should first spend some time in a laboratory where experience in making and reading tests can be obtained and should have an unquestionably reliable source of good antigen.

The Rapid Whole-Blood Agglutination Test.—The whole-blood test is made on a clean glass plate, ruled into 1- or 2-inch squares, which is placed over a white background or used as a cover for a box, the interior of which is painted white and can be illuminated. A testing box which embodies the desirable features is shown in figure 11. Similar ones are obtainable from producers of antigen or can be made at home. A white porcelain plate as advocated by some is less satisfactory. A drop of fresh blood, obtained by pricking the comb or wing veins, is transferred with a wire loop[15] (or a medicine dropper) to a square on the plate and is well mixed with the same volume of antigen. The antigen for this test is deeply colored with a violet dye. The reaction or clumping of the bacteria occurs within a few seconds to a few minutes (fig. 12). The entire procedure is carried out on the poultry farm.

The fowls are held in individual coops, numbered to correspond to squares on the glass plate in which the test is made. When the test is

[15] The loop recommended is made $\frac{3}{16}$ inch in diameter at the end of a piece of noncorrosive wire (Brown & Sharp, gauge no. 20). The loop can be made around a no. 20 steel drill or a 10-penny nail. For convenience in handling, the wire is inserted into a wood or metal handle or a cork.

completed, the fowls are retained for disposal if the reaction is positive or returned to the flock if the reaction is negative.

Some of the requirements for getting maximum efficiency are as follows:

1. The blood and antigen should be *thoroughly* mixed and spread over an area about 1 inch in diameter immediately after the blood is added.

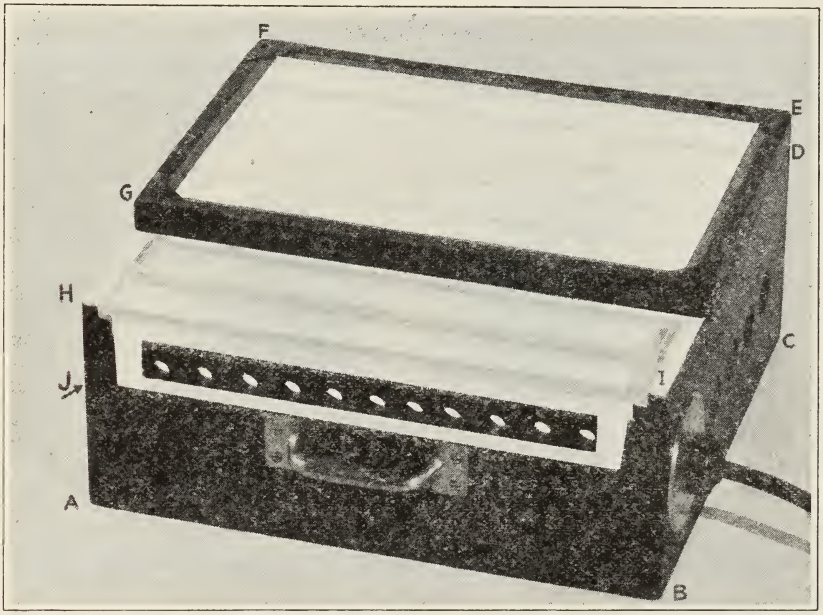

Fig. 11.—Box for making whole-blood agglutination tests. Capacity of the box is 5 glass plates, or 50 tests within the incubation chamber, and 2 glass plates, or 20 tests on the outside shelf. A thermometer should be attached to the side of the incubation chamber just beneath the test plates. The dimensions are:

AB	14¼ inches	*DE*	¾ inch
BC	13 inches	*FG*	9½ inches
CD	5½ inches	*GH*	3 ½inches

EFG, Hinged cover for incubation chamber. It protects against dust and prevents excessive evaporation.

GHI, Shelf holding two 1¾ × 13¼ inch glass test plates. Each plate contains ten 1¼-inch squares and is set ¼ inch above the metal cover of the chamber containing the lights. As soon as a test has been started in each square, the plate can be slid into the incubation chamber without raising the cover.

Compartment *GHAB* contains two 50-watt lights and is separated from the incubation chamber by a removable glass panel at the point *G*. A switch at *J* permits turning the light off and on as needed for temperature control or light for reading tests. (From Ext. Cir. 8.)

2. To insure uniformity in amounts of blood and antigen for each test, the loop should be filled so that the blood appears to bulge out and the drop of antigen is delivered with the dropper in a vertical position.

3. Between each fowl, the blood loop should be rinsed in clean water and dried on absorbent cotton or a blotter.

4. The plate on which the test is made should remain stationary. Rocking or shaking the plate may produce false reactions.

5. The plate should be level so that there is no unevenness in the depth of different portions of the mixtures of blood and antigen.

The test plate should be thoroughly washed in clean water between tests and kept free from dust by frequent wiping with a clean towel while tests are being put on.

6. The most satisfactory temperature for incubation of the tests is between 80° and 85° F. The reaction takes place more slowly at lower temperatures. Higher temperatures do not hasten the reaction and have the disadvantage of increasing the rate of evaporation.

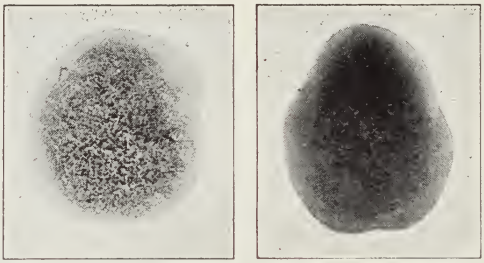

Fig. 12.—Positive (left) and negative (right) reaction to the rapid whole-blood agglutination test. (From Ext. Cir. 8.)

7. In hot, dry weather, testing should be confined to early-morning hours.

8. If the testing is done out of doors, it should be in the shade and on still days only.

9. Do not attempt to read a test after the blood-antigen mixture has begun to dry because an accurate interpretation cannot then be made.

10. Final negative readings should not be made until after 6 or 8 minutes of incubation. Some reactions develop slowly and will be missed if the final reading is made too soon. This necessitates provision of glass covers to place over the tests to protect against evaporation. These covers also protect against dust.

11. Bright, uniform light is required to facilitate reading of the tests. For this reason, artificial is preferable to natural light.

12. Fowls that give a doubtful reaction should be retested immediately. If the reaction is still doubtful, the fowl may be discarded as a reactor or a blood sample may be taken and sent to a laboratory for a check test by the tube method.

Although the conduct and reading of tests by the whole-blood method may appear to be easy to master, the ability to make accurate and

uniform interpretations of test results is acquired only by extensive practice.

The procedure to be followed in the interpretation of reactions of chickens being tested under the National Poultry Improvement Plan is as follows:

> Various degrees of reaction are observed in this as in other agglutination tests. The greater the agglutinating power of the blood, the more rapid the clumping and the larger the clumps. A positive reaction consists of a clumping of the antigen in well-developed violet flocculi surrounded by clear spaces.... A somewhat weaker reaction consists of small but still clearly visible clumps of antigen surrounded by spaces only partially clear. The interpretation of these partial reactions should be the same as that of similarly incomplete "tube method" [slow serum] agglutination reactions (i.e., strongly suspicious, weakly suspicious or questionable). Between this point and a negative reaction, there sometimes occurs a very fine granulation barely visible to the naked eye; this should be disregarded in making a diagnosis. The very fine marginal flocculation which may occur just before drying up is also regarded as negative.... As a result of more than a year of experience in testing with this antigen, it has been decided to regard as definitely positive only those reactions which appear within 1 minute after mixing the antigen and blood, while those which appear more slowly are regarded as suspicious.[16]

The opinions of research workers and livestock disease control officials differ with respect to the relative efficiency of the rapid whole-blood test and the slow serum test. Some consider the two to be of equal value. Others state that their experimental evidence shows that, as a rule, a flock repeatedly tested by the whole-blood method is likely still to contain infected birds which can be detected by the slow serum test; and, therefore, that the whole-blood test is useful for reducing the amount of pullorum disease in a flock to a low level but is not a dependable method for eradication of the disease. As a result, official testing in some states may be done by any of the three methods, in some by the rapid or the slow serum method, and in some by the slow serum test only. Official testing under the National Poultry Improvement Plan may be done by any of the three methods.

The Skin, or Pullorin, Test.—The pullorin test is made by injecting into the skin of the wattle a small amount of a fluid containing killed pullorum-disease organisms or substances extracted from the organisms. A reaction is indicated by the presence of a swelling of the wattle in which the pullorum is injected. The results of extensive trials of this test indicate that it is not a satisfactory means of detecting carriers of pullorum disease.

Necessity for Repeated Agglutination Tests.—Repeated blood tests by any of the agglutination methods are necessary for the elimination

[16] Excerpt from: United States Bureau of Animal Iudustry. The National Improvement Plan. U. S. Dept. Agr. Misc. Pub. 300:23. Revised January, 1940.

of all of the fowls in a breeding flock that harbor *Salmonella pullorum*. Some of the reasons for the failure of a single test to detect all of the infected fowls are as follows: (1) certain of the birds may have acquired the infection too recently for the production of sufficient agglutinins in their blood to cause an agglutination reaction; (2) some of the birds may become infected after the test, either from association with infected birds or from contaminated litter and soil; (3) the amount of agglutinins in the blood of a fowl that is a carrier of *S. pullorum* is variable and therefore infected fowls will sometimes fail to react to the test.

Proper Application of the Agglutination Test.—There is ample evidence to show that the intelligent and systematic application of the agglutination test is an effective and practical means for preventing pullorum disease in both chicks and breeding flocks.

Success in such an undertaking requires that *all* fowls in breeding flocks be tested frequently; that *all* reactors be promptly removed from the premises after each test; that houses, yards, and appliances be kept in a sanitary condition and be given a thorough cleaning and disinfection after *each* test; that tested stock be not allowed to intermingle with untested stock, or, better still, that tested and untested stock be not kept on the same farm; and that additions to the flock in the form of hatching eggs, chicks, or grown stock be from *known pullorum-disease-free sources*. Complete eradication of the disease from a flock will usually require several years when annual testing is the method employed. In some instances this method may serve only to keep the number of infected fowls so small that serious loss occurs infrequently in either the breeding fowls or their progeny.

More rapid progress in eradicating the disease from a breeding flock can be made, however, by retesting at intervals of a month or 6 weeks until none of the fowls react. This can be expected to require from one to five retests. Such a testing procedure should be started early enough so that one or two completely negative tests can be obtained before the hatching season begins. If the flock has been tested solely by the whole-blood method, it would be desirable to make one or more additional tests by the tube method. After pullorum disease has been eradicated in this way, the flock and its progeny should be tested annually.

Reasons for Failure to Eradicate the Disease from Flocks That Are Tested.—Cases of apparent failure of the agglutination test to accomplish much towards the eradication of infection in flocks can usually be explained by careful investigation of conditions. Some of the reasons for such failures are as follows:

1. Failure to test 100 per cent of fowls in a flock.
2. Failure to remove reactors from the flock promptly.

3. Retaining reactors on the farm for egg-producing purposes.

4. Feeding raw infertile eggs from unknown sources.

5. Failure to properly dispose of offal from reactors killed for home consumption.

6. Buying stock (eggs, chicks, adults) from flocks not free from pullorum disease.

7. Custom hatching for poultrymen who have not tested their flocks.

8. Returning birds to the flock from poultry shows and egg-laying contests without first quarantining and testing.

9. Failure to clean and disinfect the houses after the removal of reactors.

10. Mistakes in numbering blood vials.

Application of the Test to Pullets.—The agglutination test can be applied to pullets shortly before egg production has started. This practice is desirable from the standpoint of avoiding possible spread of infection through the consumption by clean birds of floor or soft-shelled eggs laid by any infected birds that may be in the flock. But unless the pullets are from a known pullorum-disease-free source, they should be tested again before their eggs are used for hatching, even though none reacted to the test.

INFECTIOUS BRONCHITIS

This disease was first recognized in 1931 in North Dakota. By 1933 it had been identified in several midwestern states and in California and become popularly known as "gasping disease" or "chick bronchitis." Its distribution is now nationwide. Probably earlier outbreaks in some sections were mistakenly taken for laryngotracheitis, which it closely resembles.

Originally the disease was believed to be confined to young chicks. More recently, however, its occurrence in flocks of adult chickens has been reported. The reports indicate, however, that infection in adults is very infrequent. One investigator states: "Of the scores of farms suffering from the ravages of this disease in baby chicks, only two reported respiratory disturbances in older fowls." Another advises: "Usually it occurs in young chicks but occasionally the disease is spread to the adults on the same farm." Numerous flocks of laying age on California farms have, during recent years, been affected with a respiratory disease which so closely resembles infectious bronchitis that it was presumed to be the same. At present, however, this is regarded as unrelated to either infectious bronchitis or laryngotracheitis. It is discussed more fully on pages 41–45.

Cause.—Infectious bronchitis is caused by one of the group of infectious agents known as filterable viruses. It is most abundant in the

exudate and tissues of the affected respiratory organs, and has also been demonstrated in livers, spleens, kidneys, and blood. In exudate, frozen, dried, and stored in a refrigerator, the virus has remained alive for 180 days. There is little definite information concerning the time the virus will remain active under natural conditions. Failure to transmit the disease by injection of exudate from dead birds suggests that the virus perishes quickly after the death of the host. On the other hand, the conditions under which some natural outbreaks have occurred suggest that the virus may remain active in a contaminated brooder house or equipment for several months. Field evidence indicates that recovered birds may become healthy carriers of the virus, but this has not been demonstrated experimentally.

Symptoms.—Gasping is the most characteristic and predominant symptom. When badly affected, the beak of the chick is pointed upward and opened wide at each inspiration. Convulsive coughing is also seen. Many chicks emit a peculiar sharp chirp. Chicks less severely affected exhibit such symptoms irregularly, but if one is held close to the ear, short, crackling sounds may be heard. Nasal discharge and swollen sinuses may be the predominant symptom, especially in chicks a few weeks old. Depression and weakness are seen in the advanced stage.

The disease occurs most frequently in chicks under 3 or 4 weeks old. It has been reported in birds as young as 2 days. The disease spreads rapidly and is apt to infect nearly all of a flock within a brief period.

The mortality is nominal in most outbreaks, the principal loss coming from growth retardation. In some outbreaks, however, the mortality may amount to from 25 to 90 per cent of those affected, and is likely to be greatest in flocks of very young chicks.

Accumulations of clear, turbid, or thick yellow mucous exudate, or caseous plugs, in the lower trachea and bronchi are the most characteristic autopsy finding. The same sort of exudate occurs in the nasal chambers when they are involved. Caseous or thick viscid mucus is occasionally seen in the larynx and air sacs.

Transmission.—The disease spreads readily from affected to healthy fowls by contact with either infected chickens or contaminated material. It is usually easily transmitted by the intranasal or intratracheal injection of exudate into the trachea or nasal passages. The source of the first infection on a farm is often obscure. Some outbreaks, however, have been traceable to hatcheries, particularly those which raise "started chicks" in batteries in the building with the incubators.

Diagnosis.—In young chicks, the symptoms and autopsy findings alone are usually sufficient for recognition of infectious bronchitis; but if there are nervous symptoms, the disease must be differentiated from

avian pneumoencephalitis. In older birds it must be differentiated from laryngotracheitis and avian pneumoencephalitis, and, when there is nasal involvement, from infectious coryza. This often requires laboratory procedures which cannot be done on a farm.

Prevention.—The only recommendations for prevention that can be given at present are the strict application of the measures of hygiene and sanitation; making as certain as possible that all new stock comes from a clean source, and that houses and equipment are thoroughly cleaned and disinfected after an outbreak. Contact between survivors of an outbreak and susceptible birds should be avoided. Recovered birds are immune, but no safe method of artificial immunization has been found. Cloacal vaccination, as used for laryngotracheitis, is not applicable because, first, it could not be applied early enough to protect very young chicks, and, second, infection of the respiratory organs can be produced by applying virus to the cloaca.

In order to eradicate the disease from establishments where continuous brooding is practiced, such as broiler plants, it is necessary that operations be suspended long enough to get rid of *all chickens* and to thoroughly clean and disinfect all pens, rooms, and equipment. Hatcheries which deal in started chicks should have separate quarters, equipment, and personnel for each operation.

No effective treatment has been found.

AVIAN PNEUMOENCEPHALITIS[17]

During 1940 a type of nervous disorder which had not been seen previously, or at least not in sufficient amount to attract attention, occurred in many flocks of growing chicks. Nearly all outbreaks of this disease either closely followed, or were accompanied by, a respiratory disease which markedly resembled and was believed to be infectious bronchitis (see preceding section). It has since been found, however, that the respiratory trouble is probably not in any respect related to infectious bronchitis. Instead, it appears to be a part of a disease of growing chicks in which both the respiratory organs and the central nervous system may be involved.

In experimental studies conducted during the present year (1942), it has been found that the disease is not confined to growing chickens but may also affect nearly mature pullets and laying chickens of any age.

[17] Less than a year has elapsed since the cause of this disease was determined and progress in the experimental studies of it thereby made possible. Consequently, knowledge concerning certain fundamental phases of it are still fragmentary and future findings may necessitate revision of some of the statements and ideas expressed here. The disease first became known as "a respiratory nervous disorder." It is now proposed, however, that this be replaced by the appropriate, descriptive, scientific name of "avian pneumoencephalitis."

Furthermore, it has been identified in two flocks of young turkeys and has been produced in young turkeys by inoculation with material from infected chickens.

Cause.—This disease, like infectious bronchitis, is caused by a filterable virus. It has so recently been identified that knowledge of its characteristics is far from complete. The virus, however, has been shown to be widely distributed in the body during the early stages of the disease, especially in severely affected chickens. It has been demonstrated in the brain, spleen, blood, lungs and air sacs, and exudate in the trachea of infected chickens. The virus has also been found in intestinal contents and in droppings collected soon after being voided. In lung tissue that has been frozen and dried or placed in 50 per cent glycerin and stored in a refrigerator, the virus has remained alive for 3 to 6 months. It grows readily in developing chick embryos, a characteristic which is a great aid in experimental studies of the disease. The conditions which are favorable to, and the time of survival of virus under natural conditions outside the body of a living chicken have not as yet been learned, but the results of very limited investigation of this point suggest that it would remain infective only a short time in a contaminated poultry house. There is, however, some experimental evidence that recovered chickens may carry the virus and therefore be dangerous to other chickens for a considerable period.

Symptoms in Chicks.—Most outbreaks of the disease in chicks have begun as a respiratory trouble, symptomatically indistinguishable from infectious bronchitis (see p. 39) but, within a few days, from a few to several of the chicks have developed symptoms of involvement of the nervous system. The respiratory phase has usually passed off within a week or two. The nervous symptoms, however, have been seen for a considerably longer time.

The respiratory symptoms are gasping, coughing and rales or rattling breathing sounds, and many of the affected chicks emit a peculiar, rapid low chirp.

The symptoms from involvement of the nervous system are quite varied in different individuals. They consist of unsteady or staggering gait, partial or complete paralysis of one or both legs, tremor or shaking of the head or whole body, and loss of coördination of action of the neck muscles, as a result of which, the head may be drawn straight back between the shoulders, downward and backward toward the breast, twisted to one side or the other, or drawn to the right or left. The chickens may walk in circles or backwards. The appetite may be unimpaired and in spite of inability to control muscular movement many chickens will manage to consume enough food to keep themselves fairly well nour-

ished. On the other hand, affected chickens may become droopy, eat little or no food, and quickly become emaciated and weak. In cases of fatal infection, the chickens are likely to become prostrated, show clonic spasms or a rhythmic twitching of the body, and go into a state of coma before death. In numerous experimentally produced cases, death has occurred in less than 24 hours after the first symptom was observed. Although some chickens have recovered from the nervous affliction, nearly all which escape death retain the symptoms indefinitely.

The most common findings on post-mortem examination of chicks have been cloudiness or thickening of the air sacs and, in many cases, also of the thin membranes of the abdominal cavity with a film of yellowish exudate and varying amounts of yellowish or clear mucus in the trachea and large bronchi. However, in many cases, particularly those with nervous symptoms alone, no visible lesions have been present. Many of the acute cases produced by inoculation have shown numerous small hemorrhages beneath the mucous membrane or lining of various portions of the digestive tract, the proventriculus in particular; but such lesions have seldom been seen in infected chicks or farms.

As was stated before, both respiratory and nervous symptoms have been present in nearly all outbreaks among chicks. The respiratory phase has often affected nearly 100 per cent of a flock, the nervous phase from 1 to 45 per cent. In a few instances, however, respiratory symptoms have been either absent or so very mild that they escaped detection. The nervous involvement tends to be more rapidly fatal in the younger chicks. The average mortality is probably between 5 and 10 per cent. A greater loss, however, may result from unthriftiness of the survivors. Most of the flocks affected have been from 3 to 10 weeks old, although outbreaks among younger and older chicks have been reported. The disease has made but a single visitation on some farms, on others it has recurred in successive broods of chicks.

Symptoms in Older Chickens.—The pneumoencephalitis virus has been isolated from a few birds affected with the disease of pullets and laying hens that is known in different localities by such names as 9-day pneumonia, California flu, and bronchitis. It is not yet known, however, that this virus is the sole cause of this type of respiratory disease. Although the symptoms resemble both infectious bronchitis and infectious laryngotracheitis, this disease appears to be unrelated to either. It has occurred in many flocks that had been immunized to laryngotracheitis and a number of attempts to transmit it by the procedures successfully employed for the transmission of infectious bronchitis and laryngotracheitis have failed. This disease is characterized by a sudden onset and extremely rapid spread through a flock. The loss from death has usually

been nominal. The egg production of laying flocks, however, may practically cease within a week and not return to a profitable level until 1 or 2 months later. Affected chickens cough, gasp, and manifest other symptoms of difficult respiration. They may also become listless and lose interest in food. Improvement is likely to be seen within a week or 10 days. At autopsy varying amounts of clear or cloudy liquid or viscid mucus are found in the trachea. Exudate in the trachea of the thick, yellowish and bloody nature which characterizes laryngotracheitis has rarely been seen.

Another type of disease of laying chickens, with which the pneumoencephalitis virus has recently (August, 1942) been identified, was manifested as follows: The first effect of the disease was to cause a decrease in appetite, especially for mash. Within 2 or 3 days, depression or dullness of from a few to several of the chickens was seen. During the following few days more chickens showed these symptoms and, in some outbreaks, by the end of a week practically all the chickens in a pen would be sitting about on the roosts or floor and eating little or no food. Egg production has uniformly decreased rapidly, sometimes ceasing entirely. At the beginning of some of the more severe outbreaks, a large number of floor and yard eggs, many with soft shells, were laid. Some coughing has usually been heard, but respiratory symptoms were not prominent. In addition to the above, a variable number of the chickens developed nervous symptoms like those described for chicks (p. 42). Affected flocks have begun to improve within a week or 10 days after depression was first seen and have quickly regained normal appearance. Egg production, however, has increased more slowly. The mortality has varied from a negligible number to as high as 22 per cent of the flock. On some farms on which the disease has appeared for the first time, it has progressed from pen to pen until the whole population, regardless of age, has become infected. Lesions by which the disease could be identified have not been found on post-mortem examination of dead chickens.

Transmission.—Experimentally, the disease has been transmitted by injecting fresh blood or tissue or exudate of the respiratory organs, brain, or spleen of infected chickens into the nasal passages, trachea, air sacs, breast muscle, brain, abdominal cavity, crop, or bursa of Fabricius or under the skin of normal chickens which ranged in age from a few days to more than two years. Transmission by pen or cage contact has also been readily accomplished. The means by which the infection may be first introduced into a farm, however, are still too indefinite to be discussed here. Outbreaks in chicks in some instances have been preceded by the occurrence of the respiratory disease in older chickens on the farm. In some cases the situation has been reversed.

Diagnosis.—A combination of respiratory and nervous symptoms in a group of growing chicks is at present considered sufficient for fairly certain recognition of the disease. Laboratory procedures must be employed to differentiate it from infectious bronchitis, however, if only a very small percentage of a flock shows nervous symptoms. The disease does not produce any constant and characteristic lesions which enable one to recognize the disease by post-morten examination. Yellowish-gray cloudiness of the ordinarily clear membranes which form the air sacs is the most frequent lesion encountered, but this, in many cases, is absent or indefinite.

The respiratory disease of older chickens must be differentiated from infectious bronchitis and infectious laryngotracheitis, and this usually will require laboratory procedures. It is likely not to be laryngotracheitis, however, if none or but a few of the affected chickens die, if the flock has recently been vaccinated for laryngotracheitis, or if no bloody mucus is coughed up or is found in the trachea by post-mortem examination. These, however, are not certain differential factors. Symptoms in laying flocks, as described on page 44, are highly suggestive but are not diagnostic of infection with the pneumo-encephalitis virus, and, therefore, laboratory procedures are required for positive identification of even such cases.

Control and Prevention.—A measure that appears worth while for flocks of chicks or older chickens affected with pneumoencephalitis consists in endeavoring to stimulate the birds' food consumption by some modification of the feeding method, such as giving the mash in meals instead of keeping it before the birds all of the time, supplying extra milk products, and giving an extra daily feeding of fresh tender greens, moistened mash, and rolled barley soaked in milk. This may reduce the number of unthrifty birds in flocks of chicks and shorten the period of low egg production in laying flocks.

By careful observance of precautions against carrying the infection from one pen to another, it may be possible to confine the disease to one or two pens. The attendant should always take care of the affected pens last, should have coveralls which are worn only in the infected pens, should thoroughly wash his hands, using plenty of soap, when he has finished caring for an infected pen, and should have separate utensils for use in the infected pens. The chickens should be kept inside, and cases showing nervous symptoms should be destroyed as quickly as they appear.

Houses and equipment should be thoroughly cleaned and disinfected after an outbreak and the survivors probably should be segregated from other chickens which are on the farm at the time or added later. To

eradicate the disease from pullet-raising or broiler plants on which brooding is continuous, it is likely to be necessary to suspend operations until all chickens can be disposed of and the pens, rooms, and equipment thoroughly cleaned and disinfected.

INFECTIOUS AVIAN ENCEPHALOMYELITIS (EPIDEMIC TREMOR)

This disease of the nervous sytem of chickens has been present in northeastern states since 1930. It was identified in California for the first time in December, 1941, in chicks of the New Hampshire breed. Since then the disease has occurred in a few additional flocks of New Hampshires and White Leghorns as well. It was at first termed "epidemic tremor" because a prominent and striking symptom is tremor or rapid shaking of the head. This name is not strictly applicable, however, because subsequent studies have revealed that less than half of affected chickens show the tremor symptom.

Cause.—The disease is caused by a filterable virus. The brain of affected chicks is the most reliable source of virus, but it has been demonstrated also in the spleen and liver.

Symptoms.—The first symptom to appear is ataxia, or unsteadiness and inability to control movements of the legs, which becomes progressively more pronounced. Many chicks lose the ability to stand, and sit and walk on their hocks. Some become unable to move at all and soon die. In a variable number of the chicks, the ataxia may be followed in from a few to several days by the appearance of a rapid tremor of the head. This becomes more prominent when the chicks are disturbed. Death may take place soon after the onset of symptoms, or after a more protracted illness. A considerable number of the affected usually survive, however, and the majority of these retain the nervous symptoms. In fact, the number of affected chicks which die from being trampled by the healthy ones may be greater than that from the direct effects of the disease. A flock usually becomes affected at the age of 1 to 6 weeks. New cases can continue to develop for several weeks. The total number of chicks to contract the disease ranges from 5 to 50 per cent or more of the flock. On some farms, successive hatches of chicks have become infected, on others the disease has appeared very irregularly or only once.

Transmission.—Some who have studied the disease report having obtained both field and experimental evidence that the infection is transmitted through the egg from the hen to its progeny and that this is a probable means of introducing the disease to a flock hitherto free from it. This view, however, is not supported by the experimental findings of all investigators.

Spread of the disease by direct contact has been observed both in farm

flocks and in experimental groups of chicks. Experimentally the infection has also been transmitted by injecting brain tissue of diseased chicks into the brain or nasal passages of healthy ones.

Diagnosis.—It is not possible to recognize avian encephalomyelitis in chicks from symptoms alone. Neither can this be done by post-mortem examination of dead chicks on the farm because the disease produces no lesions which are visible to the unaided eye. Therefore a diagnosis can be made only at a laboratory by microscopic examination of the brain and inoculation of healthy chicks.

Chicks fed a diet deficient in vitamin E develop symptoms much like those of avian encephalomyelitis and therefore this possibility must be considered in any outbreak. Although poultry rations are not usually lacking in vitamin E, it appears that the vitamin may be destroyed or inactivated when fish oil is used at high levels or has become rancid. The symptoms of avian encephalomyelitis also are similar to and must be differentiated from the nervous phase of pneumoencephalitis in chicks, described in the preceding section.

Control and Prevention.—Affected chicks should be immediately taken out of the flock and either destroyed or kept separate and sold for meat as soon as they reach broiler size. This may both check the spread of the disease and remove from the farm possible reservoirs of infection of other chickens.

Since the possibility of transmission through the egg exists, it would be inadvisable to purchase hatching eggs or chickens of any age from a flock in which the disease is or has been present. For the same reason, survivors of an outbreak might be considered unsuitable for breeders.

INFECTIOUS LARYNGOTRACHEITIS

This disease was previously designated as "infectious bronchitis," but, since studies of the pathology of the disease have shown that it affects principally the larynx and trachea, the more correct term, *infectious laryngotracheitis,* has been adopted. The disease has become widespread and of major economic importance in the United States and Canada, but is not known to have occurred elsewhere in the world, except in Australia, where it was identified in 1935. Reports of its occurrence in England, Germany, and Spain are of doubtful authenticity.

Cause.—The disease is caused by a type of infectious agent known as a "filterable virus," which is found principally in the exudate that forms in the larynx and trachea. Exudate from an infected fowl, when dried and kept under suitable conditions, has remained capable of producing the disease for more than a year.

It has been shown experimentally that the virus is quickly destroyed

by exposure to moderate temperature (55° to 75° C), does not survive longer than 90 days at room temperature, may be killed by exposure to direct sunlight in 7 hours, survives in the body of a dead chicken only until decomposition of the tissues begins, and is readily destroyed by a 3 per cent solution of cresol disinfectant and a 1 per cent solution of sodium hydroxide (lye). The importance of these findings is that they indicate that the virus is not likely to survive in contaminated poultry houses or equipment from one season to another and that either a cresol disinfectant or a lye solution is an efficient disinfectant for use on poultry farms in which the disease has occurred.

Fig. 13.—An advanced case of infectious laryngotracheitis, showing attitude during expiration. (From Ext. Cir. 8.)

It has been found that chickens may carry the virus in their tracheas for as long as 16 months after recovery from the disease. Such "healthy carriers" can infect susceptible chickens with which they become associated and are the probable source of the infection causing annual outbreaks among the young stock on many farms.

Symptoms.—The predominant symptoms of the disease are coughing and gasping for breath. The affected fowl assumes a sitting position, with the neck drawn in, the beak pointed downward, and the eyes closed (fig. 13). At each inhalation, the head is thrown forward and upward, with the beak opened (fig. 14) and the intake of air is accompanied by a loud wheezing sound. Spasmodic exhalation or coughing is frequent and often results in the expulsion from the trachea of a mass of clotted blood. Examination may reveal the larynx to be nearly filled with bloody or yellowish, thick mucus or with caseous material, while in other cases, in the live bird, the cause of respiratory difficulty is not visible. Rarely, the disease may also involve the eyes and nasal passages.

The onset of the disease is sudden and as a rule the spread through a flock is very rapid. An outbreak usually causes a mortality of from 5 to 25 per cent or more of a flock during a period of from 2 to 4 weeks. The disease then disappears about as suddenly as it began. Laying flocks usually almost cease production and do not again attain a normal production rate for 1 or 2 months. Occasionally the spread is slow and evidence of the disease may exist in a flock for weeks.

Fig. 14.—Same fowl as in figure 13, showing attitude during inspiration. (From Ext. Cir. 8.)

When there is more than one flock on a farm, it may attack one flock after another. Recurrences of the disease in the same flock do not take place, however, unless only a small number of the fowls were initially infected, the reason being that recovered fowls are permanently immune.

The majority of the fowls that die do so within 1 or 2 days after symptoms appear. A large percentage of affected fowls that survive the first 2 days of sickness recover. Not infrequently the value of the eggs lost by the decrease in production exceeds that of the fowls which die.

On autopsy, all organs are apparently normal with the exception of the larynx and trachea. These organs are usually wholly or partially filled with mucus and clotted blood and their linings inflamed. In some cases of 3 or 4 days' duration, the mucus in the larynx becomes caseated. Occasionally caseous mucus is found extending the entire length of the

trachea. In some birds, the amount of clotted blood present exceeds that of mucus. The collection of mucus is sometimes confined to the lower portion of the trachea, the upper portion of the trachea and the larynx being entirely free. The lungs appear normal except for small areas of congestion. Death in most cases appears to be due entirely to asphyxiation, which occurs when the larynx or trachea becomes filled with mucus or clotted blood.

Birds Affected by the Disease.—Laryngotracheitis has occurred or been produced only in chickens and pheasants. Domesticated ducks, turkeys, pigeons, and wild and free-flying species of birds, including sparrows, crows, starlings, doves, and quail, have been found refractory and so, too, have rabbits, guinea pigs, and white rats.

Diagnosis.—Many outbreaks of laryngotracheitis can be readily recognized by the characteristic symptoms and autopsy findings, and the rapidity of spread of the disease through a flock. Almost identical characteristics, however, are presented by infectious bronchitis of chicks and the similar disease in adult chickens (p. 43). A respiratory disease of this character in young chicks, however, is not likely to be laryngotracheitis, for this disease has rarely been seen in such young birds. A respiratory disease of older chicks, which has symptoms of laryngotracheitis but which is accompanied by few or no deaths and the collection of clear or slightly cloudy mucus, instead of yellowish or bloody, in the trachea is likely to be infectious bronchitis. In many cases, however, a positive diagnosis cannot be made on a farm, but must be based upon the results of transmission, cross-production, and other tests at a laboratory.

In some outbreaks of a severe type of coryza, many of the affected fowls have tracheal involvement, and coughing and gasping are prominent symptoms. The diagnostic question in such cases is to determine whether the fowls have coryza alone or are affected with both coryza and laryngotracheitis. Laboratory procedures are usually necessary to provide the answer.

Vaccination.—Vaccination procedure is based upon the discovery that the mucous membrane of the cloaca and bursa of Fabricius (fig. 15) is susceptible to the virus of laryngotracheitis, that infection of these parts does not cause a systemic disturbance or spread to the respiratory tracts, that the cloacal infection subsides in about 7 days, and that the fowl is thereafter immune to infection of the respiratory tract.

The vaccine consists of exudate taken from the trachea of artificially infected chickens or, as in fowl pox (fig. 23, p. 22), the chorioallantoic membrane of chicken embryos on which the virus has been propagated. The two types of vaccine are labeled by producers respectively as "chicken origin" and "egg propagated" or "chick-embryo origin." The virus-

bearing exudate or embryo tissue, after removal, is dried and finely powdered. This treatment, when properly done, does not reduce the virulence of the virus. This vaccine, therefore, will cause severe infection

Fig. 15.—(1) Bursa of Fabricius; (2) large intestine; (3) cloaca; (4) vent, or cloacal lips. (From Ext. Cir. 8.)

if it is introduced into the respiratory passage of a susceptible chicken. *It must be handled carefully and strictly in accordance with instructions.* For use in vaccination, the powdered virus is suspended in a solution of glycerin.

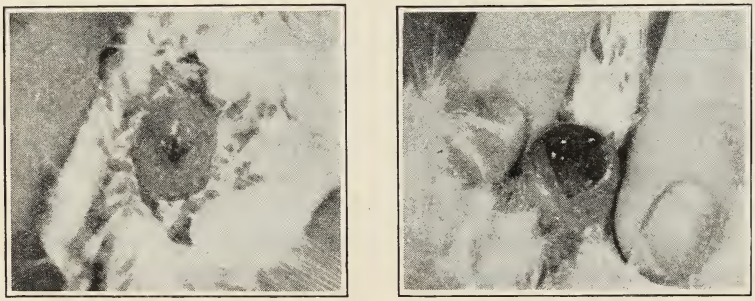

Fig. 16.—Take from laryngotracheitis vaccination: left, swelling of the cloacal lips; right, reddening of the cloacal mucous membrane. (From Ext. Cir. 8.)

The vaccination procedure consists in brushing the vaccine onto the cloacal mucous membrane; the cloacal lips are forced open to expose the membrane which is then brushed until redness or even bleeding is produced. Another, although little-used, method of vaccinating chickens under the age of 5 to 5½ months consists of injection of the vaccine directly into the bursa of Fabricius through a blunt, slightly curved hypodermic needle. The resultant take, or infection of the mucous membrane (fig. 16), consists of swelling of the cloacal lips, redness and swell-

ing of the mucous membrane, and the presence on its surface of mucus, often yellow and flecked with blood. A take usually reaches maximum intensity on the fifth day and by the seventh day has nearly disappeared. In contrast to survivors of respiratory infection, the virus does not persist in the cloaca or bursa of Fabricius after the reaction subsides.

The birds should be examined on the fifth day after vaccination, and any that do not show a definite take should be revaccinated immediately. If not revaccinated, the birds without a take can be expected to acquire severe respiratory infection from the virus eliminated by the birds with takes. Failure to obtain vaccination takes may be due either to faulty administration or to impotence of the vaccine. The potency of vaccine is likely to be adequate when it leaves the producing laboratory but can be easily reduced by exposure to warmth during transit to and after reaching the farm. It should be protected against warmth even while it is actually being used for vaccination.

No special additional after care is required.

Chickens can be vaccinated at any time after they reach the age of 6 weeks, but preferably before they begin to lay. All instructions furnished with the vaccine should be faithfully obeyed.

Vaccination is indicated:

1. For the prevention of the spread of the disease on a farm after it has appeared in one pen. In such cases the noninfected pens should be treated first.

2. For young stock on farms where the disease has occurred in the past and where survivors of a previous outbreak remain.

3. For susceptible fowls which are added to a flock in which the disease is or has been present; or for a susceptible flock to which known survivors of the disease are to be added.

4. For healthy flocks, either already existing or newly established in a congested poultry district in which the disease is prevalent.

Vaccination is not indicated:

1. As a preventive measure in a flock not previously infected unless it is located in a congested poultry district in which the disease is prevalent.

2. For the control of an outbreak of any respiratory disease not definitely diagnosed as laryngotracheitis.

3. For a portion of the susceptible birds on a farm, unless the birds not vaccinated are too young, are well segregated, and are to be vaccinated as soon as they reach suitable age.

4. For use by persons unfamiliar with the hazards attendant upon the use of a virulent live-virus vaccine.

Prevention.—The strict application of the principles of hygiene and sanitation (p. 22) should be adequate to prevent the introduction of the

disease into a flock not located in a poultry district. Particular attention should be given to the source of added stock and any article that is used in poultry houses.

Prevention of recurrence of the disease on a farm after an outbreak without annual vaccination has been accomplished by the following procedure:

1. Removing from the premises all birds which have had or have been exposed to infectious laryngotracheitis.
2. Thoroughly cleaning and disinfecting the outer clothing of the attendant and all buildings and equipment used for housing and care of the condemned chickens.
3. Leaving the houses and equipment unused for at least 3 months after they have been cleaned and disinfected.
4. Retaining chicks for restocking on the premises provided they are well separated from the condemned birds and are definitely known to have escaped infection.
5. Securing new stock (preferably as baby chicks) from absolutely clean flocks.

These measures would be of questionable effectiveness in poultry districts in which infectious laryngotracheitis is prevalent.

Treatment.—No medicinal treatment for individual birds or a flock has been found to have sufficient merit to warrant its use; in fact, dropping chemicals into the trachea or spraying them over fowls on the roost may increase the respiratory distress of affected birds. The reason that many of the chemicals that have been tried as a treatment for the disease have appeared to be beneficial is that a large percentage of infected birds recover spontaneously without treatment.

When difficult breathing is due to stoppage of the larynx and upper trachea, quick relief and recovery can usually be obtained by careful removal of the material with forceps. This is the only type of treatment that has been found worth while.

INFECTIOUS CORYZA, OR COLDS

Coryza (the scientific name for nasal catarrh or cold in the head) is probably the most commonly occurring and widespread of all diseases of poultry. In the past, environmental conditions, such as damp weather, sudden changes in temperature, crowding, and drafty or poorly ventilated houses, were regarded as necessary predisposing or even primary causative factors, and the microörganisms present in the nasal chambers of affected chickens were presumed to be only secondary factors. In 1933 and 1934, however, several investigators, widely scattered and working independently, succeeded in isolating from the nasal exudate a species

of bacteria which, when injected into the nasal passages of healthy chickens, would reproduce the symptoms of the natural disease. This type of respiratory infection, to which the name "infectious coryza" has been given, is very prevalent on poultry farms.

Cause.—The bacterium named *Hemophilus gallinarum,* the fowl-coryza bacillus, is considered to be the primary cause of infectious coryza. This organism is similar in form and growth requirements to the bacillus associated with influenza of man and certain domesticated animals. It grows sparsely in cultures; this may be why it was not identified earlier.

Fig. 17.—Chickens with infectious coryza.

The bacillus can be readily demonstrated microscopically in exudate and obtained in culture during the early stages of the disease. In older cases, however, it is difficult or impossible to identify the organism because of the numerous other bacteria which are present. Consequently, to identify a field case as infectious coryza, it is often necessary to inoculate chickens with the nasal exudate in order to secure for examination a case which has just begun to show symptoms.

The coryza induced by inoculation with a culture is usually of shorter duration than that from natural infection or that induced by inoculation with exudate from a field case. This has raised the question of whether the natural disease is caused by *Hemophilus gallinarum* alone or in combination with another causative agent. On this point the opinions of research workers differ. Some believe that the relative mildness of disease produced by inoculating chickens with cultures may be due to reduction in the virulence of the organism by cultivation in an artificial medium. Two other organisms, one termed "coccobacilliform bodies"

and the other *Shigella nasalis,* have been isolated and are said to be capable of acting in conjunction with the fowl-coryza bacillus to increase the severity of the infection produced by the latter alone. From the preceding it is seen that the cause of infectious coryza is not as yet fully understood.

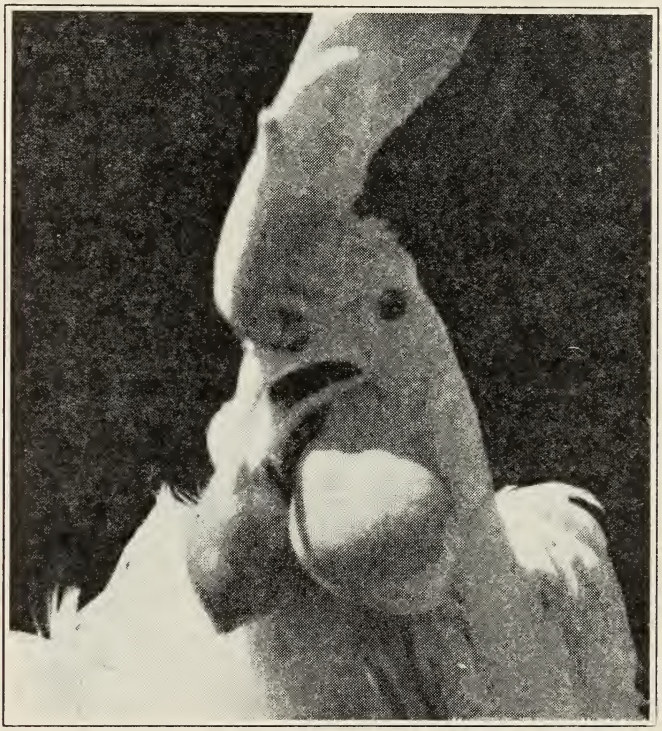

Fig. 18.—Edema of the wattles produced by injection of a culture of the fowl-coryza bacillus.

In addition to the above, the fowl-cholera bacillus may, under certain circumstances, become localized in the nasal passages of chickens and produce a coryza of the same type as that under discussion. This disease is dealt with in the same way.

Symptoms.—Infectious coryza has shown a remarkable variability in its severity and harmfulness to a flock. The mildest type of the disease occurs as a simple coryza, the only symptom of which is a nasal discharge, either persistent or of short duration. Indications of systemic effect, such as droopiness and diminished appetite, are absent.

In the severer types of the disease the coryza is complicated by other manifestations. One frequently seen is edema or puffiness (fig. 17) of the face which may extend, especially in males, to the wattles (figs. 18,

19). The latter condition, however, often results from other causes (see p. 75) and is not indicative of infection with the fowl-coryza bacillus unless accompanied by other symptoms of coryza. Other complications are swelling beneath the eyes and involvement of the trachea, bronchial tubes, and even the air sacs.

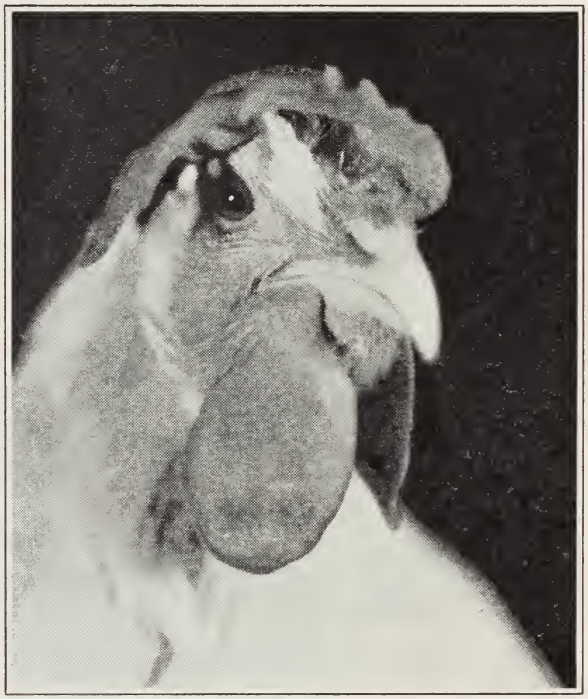

Fig. 19.—Edema of wattle from natural infection with coryza. The chicken also has swelling under the eyes, which is not clearly shown in the photograph.

The heaviest losses occur when a large number of the affected chickens have involvement of the trachea and bronchi. In such cases, coughing and gasping are prominent symptoms and there are numerous deaths from suffocation, which gives the disease a close resemblance to laryngotracheitis. In addition the fowls are depressed, have diminished appetite, and become progressively emaciated.

The course of infectious coryza in a flock is usually prolonged, extending over a period of several weeks or months. The resultant mortality varies from a negligible number to more than 50 per cent of a flock. In an extreme case the disease persisted in a flock of 2,500 pullets from May to December and ultimately caused the destruction of all the birds. In many outbreaks, however, the loss from decreased egg produc-

tion and the development of worthless culls exceeds that due to immediate mortality.

The mild type of coryza is extremely insidious. The damage which it does may seem to be so slight that the flock owner disregards its presence even though a relatively large number of birds may be affected. The adverse effect on egg production, however, is likely to be considerable, despite the fact that the birds look well, eat well, and few are dying. But the greater danger lies in the possibility of a change from a mild to severe type of disease at any time, with consequent heavy loss. Such a change can readily be brought about by rapid passage of the disease from one chicken to another at a laboratory and has been observed on farms.

Chickens which recover may carry the infection for an indefinite time and may again develop symptoms. Consequently, after a flock has been infected once, it is likely to always contain some healthy carriers or mildly affected birds from which the disease is transmitted to others. This is believed to be the means by which the infection is perpetuated on a farm so that it occurs in pullets year after year. Transmission takes place readily through direct contact between infected and healthy birds. It can also be transmitted by indirect contact, such as by contaminated material or clothing of the attendants, but the exact agency through which this takes place is often obscure.

Species Susceptible.—Pigeons have been found refractory to infectious coryza. The disease has been transmitted to turkeys and the resultant disease was of the same character as natural cases of coryza and sinusitis (swell head) in this species of bird. *Hemophilus gallinarum*, however, has not been identified in natural cases of sinusitis in turkeys.

Diagnosis.—Experience has shown that any respiratory disease on a California farm which has a nasal discharge as a constant symptom, affects several chickens at a time, and persists in a flock over a period of weeks or months, and especially one which affects pullets year after year, is likely to be infectious coryza. It is always well, however, to have a field diagnosis checked at a laboratory to determine whether the disease can be transmitted by inoculation of healthy chickens with nasal exudate and whether the fowl-coryza bacillus is present. This is particularly desirable for identification of the mild type of the disease. When the symptoms consist of coryza and edematous swelling of the face or wattles, laboratory procedures are necessary to determine whether the infection is with the fowl coryza or the fowl-cholera bacillus. When symptoms of tracheal involvement—that is, coughing and gasping—in addition to nasal involvement are present, laboratory procedures are required to determine whether the disease is coryza alone or a combination

of coryza and infectious laryngotracheitis if in half-grown or laying birds, or coryza alone or in combination with infectious bronchitis if in chicks. It should be apparent from the preceding that definite differentiation of respiratory diseases of chickens in many cases is a difficult problem.

Prevention.—Measures of hygiene and sanitation (p. 22) conscientiously applied should ordinarily be adequate to prevent the introduction of infectious coryza on a poultry farm. If a farm is in a congested poultry district in which the disease is prevalent, prevention by sanitary measures is more difficult but is more likely to succeed if the chickens are confined in the houses. Under any circumstances, it is particularly important that additions to the flock be made only by hatching eggs or day-old chicks.

The prevention of recurrences of the disease after it has once appeared on a farm requires complete and permanent separation of survivors of infected flocks from all other chickens. This may be accomplished either by segregating the survivors in a separate house on the farm or by depopulating, which entails removing all survivors of an infected flock from the premises.

In segregation, the infected fowls should be confined in a house as far removed from others as possible and be kept thus separated as long as any of them remain. Extreme care should be taken to see that none of them escape into pens occupied by others. It is preferable that their caretaker does not also care for other fowls; but, if this cannot be done, the work should be planned so that they are always the last to receive attention. A separate set of utensils should be provided. This procedure has been only partially successful on most of the farms on which it has been tried, and on some it has failed utterly.

For elimination of infectious coryza by depopulating, three plans have been successfully used:

1. Selling all of the condemned flock as market poultry, and restocking the farm with chicks.

2. Moving the condemned flock to other premises, usually rented for the purpose, and restocking with chicks. The fowls removed are maintained in their new location until all are disposed of in the usual manner.

3. Rearing the chicks for replacement until the birds are about ready to lay on other premises where they will not be exposed to coryza. The condemned flock is then marketed and replaced by the healthy pullets.

In plans 1 and 2 the chicks for replacement may be on hand as long as 2 months before the older chickens are removed if the brooder houses are well separated from other poultry houses and separate attendants and utensils are provided for the chicks. When plan 2 or 3 is adopted,

it is highly desirable to have separate attendants and equipment for each of the two flocks.

The vacated houses and the equipment can be used again as soon as they have been thoroughly cleaned and disinfected. With any one of the three procedures, the new flock can be expected to remain free from infectious coryza until it is reintroduced from the outside through nonobservance of sanitary precautions (see p. 22), such as adding pullets, breeding cockerels, or adult chickens to the flock. To obtain sustained success from depopulation of a poultry farm in close proximity to others, exceptional precautions must be taken. Under such circumstances, the clean flock should be confined to houses and wire netting, preferably 1-inch mesh, enclosed sun porches; and the areas of the farm on which poultry houses are located should be well fenced to keep out any stray chickens from neighboring farms. Depopulation may appear impractical because of the cost, but it will be found less expensive than the loss by death, retarded development, and reduced egg production that results when each year's crop of pullets becomes affected with coryza.

Vaccination with avian mixed bacterin has not proved effective for the prevention of infectious coryza.

Control and Treatment.—In outbreaks of infectious coryza, some benefit may be derived from the isolation of affected fowls until they have recovered. If begun early and conscientiously continued, the spread of the infection may be so retarded and the severity so reduced that the mortality will be small. Little benefit can be expected from this procedure, however, unless it is started before many of the birds have become infected. The recovery of sick fowls may be hastened by irrigating the nasal passages with liberal amounts of a dilute hypochlorite solution or other nonirritating disinfectant. For this, a poultry nasal irrigator is desirable. Severely affected fowls had better be destroyed at once.

Claims that increasing the vitamin A in the feed for a flock with infectious coryza will lessen the severity and shorten the course of the disease have not been substantiated by the results of experimental studies. Such studies have shown that the resistance of chickens to the disease may be enhanced by giving them amounts of vitamin A greatly in excess of normal requirements for a few weeks before they are exposed. No beneficial effect was demonstrated, however, when the feeding of an excess of the vitamin was started at the same time as exposure to the disease or after symptoms had appeared.

When the appetite of the flock is impaired, an endeavor should be made to tempt the chickens to eat more by some modification of the feeding practice. They may, for example, be offered a daily light feed of moist mash, rolled barley soaked in milk, and fresh succulent greens.

Sick chickens are not benefited by vaccination with avian mixed bacterin, by medication of the feed and drinking water, or by having chemicals sprayed over them when they are on the roosts.

Other Comments.—It should not be inferred from the foregoing that all coryza in chickens is caused by the infectious agents mentioned nor, indeed, that all is infectious. Not infrequently failure results from attempts to show that coryza, affecting an individual or a few birds of a flock, is infectious and transmissible. A coryza of a noninfectious type, however, is not likely to be persistent, nor is it likely to affect many birds.

It is probable that an exaggerated importance has been attached to exposure of chickens to cold and dampness or faulty housing conditions, such as draughtiness, poor ventilation, and overcrowding, as causes of coryza. It is, nevertheless, well to assume that such unfavorable conditions may be responsible for the disease, and that infectious coryza would be more damaging under such circumstances. Therefore, it is advisable to keep chickens inside during wet weather, and, whenever coryza is seen, to correct at once any possible faults in housing conditions that can be found.

In mild attacks of coryza, the possibility of vitamin-A deficiency should be considered because a nasal discharge is a common and early symptom of deficiency of this vitamin (see p. 100). This could occur without being suspected if vitamin A was being supplied principally by alfalfa meal and the quality of this ingredient of the mash happened to be poor.

Any case of coryza should be regarded as potentially dangerous and the infected bird removed from the flock. If more cases appear, steps should be taken to ascertain if infectious coryza is present. By so doing the disease may possibly be headed off before it gets out of hand.

FOWL POX

Pox in birds has a world-wide distribution and a variety of bird hosts. It is of greatest economic importance as a disease of chickens and turkeys but is seen occasionally as a natural disease of pigeons and canaries. The several cases of the disease in wild birds that have been seen in California were apparently of chicken origin. Pox of chickens can affect a wide range of domesticated and wild birds, exceptions being pigeons, which are not affected by the chicken strain of the virus, and water fowl. Outbreaks are most prevalent during the fall and winter but may occur at any time of the year.

Cause.—Fowl pox is one of the group of diseases caused by the type of infectious agents known as filterable viruses. The virus is present in large amounts in the lesions which form on the skin. It is present in

blood and internal organs to a much less extent or not at all. This virus survives for a long time on poultry farms and is resistant to disinfectants. Consequently, it is very difficult to completely remove or destroy the infective material remaining in a poultry house after an outbreak of the disease.

Fig. 20.—Fowl pox in a cockerel; natural infection. (From Ext. Cir. 8.)

Symptoms.—The characteristic lesions are small wartlike growths on the comb, wattles, and skin of the face (figs. 20, 21). They may also occur on the feet and legs and any unfeathered area of the skin of molting birds. The lesions begin as minute, smooth, yellow eruptions. Their size rapidly increases, their surfaces become roughened and dry and the color changes to dark brown. Within 2 or 4 weeks a dry scab forms, loosens, and drops off. Individual lesions often join so that a large area becomes solidly covered. Skin lesions are frequently accompanied by the formation of masses of soft, yellow, cheesy material or canker on the mucous membrane of the mouth and eyes.

The effect of the disease on an individual is proportional to the extent of the lesions. A few pocks on the skin dry and drop off without any

appreciable effect on the general health. Extensive skin lesions and canker, on the other hand, are more lasting and are accompanied by depression, loss of appetite, emaciation, and, in many cases, eventual death. Canker may interfere with consumption of feed or cause loss of eyesight or death from suffocation.

Outbreaks of fowl pox vary greatly in severity. In some cases the disease may be restricted to a few birds of a flock and cause little damage, while in other flocks the lesions may be very severe; the disease may spread through almost the entire flock and cause a great loss, both from

Fig. 21.—Fowl pox in a baby chick; natural infection. (From Ext. Cir. 8.)

decreased egg production and from the death of the infected fowls. The mortality is greatest when a large percentage of the infected fowls have canker in addition to lesions on the skin. In many outbreaks in laying flocks, the loss from decreased production of eggs is greater than from death of chickens.

Chickens that recover from pox are solidly immune against reinfection, usually for life. Recovered fowls may be virus carriers, but for how long and in what manner has not been definitely shown. Nevertheless, there is enough field evidence to indicate that it is unsafe to mix susceptible chickens with those that have had fowl pox.

Diagnosis.—The appearance of fowl-pox lesions about the head is usually so characteristic that one has little difficulty in recognizing the disease. Skin lesions in other locations, and occasionally on the comb as well, are less typical, and inoculation of susceptible chickens is necessary to positively identify them. Doubtful cases should be submitted to a diagnosis laboratory.

Canker of unknown origin or resulting from irritation by particles of litter or other foreign material which may become lodged in the eye or the cleft in the roof of the mouth is often present in a few birds in any flock. These are not likely to be due to fowl pox unless skin lesions of fowl pox are also present.

Preventive Vaccination.—Vaccination of growing chickens to protect them from fowl pox after they mature has been practiced for more than twenty years and is now done routinely on many poultry farms.

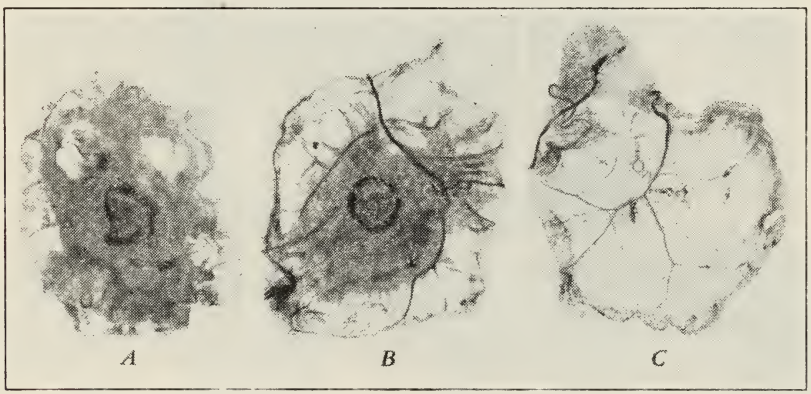

Fig. 22.—Chorioallantoic membranes of chick embryos infected with pox virus: *A*, membrane infected with pigeon virus; *B*, membrane infected with chicken virus; *C*, normal membrane. In making vaccine, the thickened area of the membrane is dried and finely powdered.

The vaccine consists of lesions taken from the comb of artificially infected young chickens or from the chorioallantoic membrane (fig. 22) of chicken embryos on which the virus has been propagated by introducing it through a window cut in the eggshell (fig. 23). The source of virus in vaccine is indicated on the label of the container by the words "chicken origin," "chick-embryo origin," or "egg-propagated." The virus-bearing tissue is dried and powdered. For use in vaccination, the powdered virus is mixed with sterile water, physiological salt solution, or a solution of glycerin. This treatment does not influence the virulence of the virus. The vaccine, therefore, is capable of causing severe fowl pox and must be handled carefully and strictly in accordance with instructions.

Vaccine prepared from pigeon strains of pox virus is also available. This is to be used only when chicken-virus vaccine is contraindicated (see p. 69).

Methods of Vaccination.—Chickens may be vaccinated with chicken-virus vaccine by either of two methods, the feather follicle or the stick method; with pigeon-virus vaccine, by the former only.

The *feather-follicle method* consists in applying the vaccine with a small brush to a few feather follicles on the leg from which the feathers have been plucked. When using chicken-virus vaccine not more than four or five follicles should be exposed; for pigeon-virus vaccination the number of follicles may be increased to from ten to twenty.

Fig. 23.—Incubating egg in which chicken-pox virus is propagated. The seed virus is deposited on the chorioallantoic membrane which surrounds the chick embryo and the window is then rimmed with melted paraffin and sealed with cellophane.

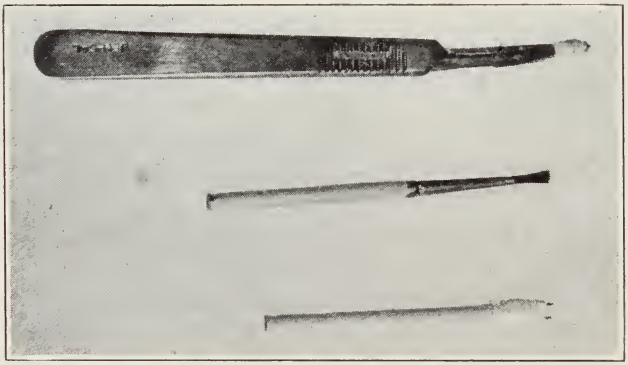

Fig. 24.—Types of instruments in common use for vaccinating against fowl pox. (From Bul. 613.)

The *stick method* consists in making one or more pricks in the skin with a sharp-pointed instrument immediately after it has been moistened with vaccine.

A common place for vaccination by the stick method is the outside of the upper end of the drumstick or on the breast. The feathers should be parted to expose the skin before the "stick" is made. The points of the vaccination instrument should be shortened to about ⅛ inch by wrapping with tape or thread (fig. 24). This prevents too deep penetra-

tion of the points into the muscle tissue and also helps to insure introduction of vaccine into the punctures through the skin.

Another vaccination site is the web of the wing. Vaccination here is done by piercing both layers of skin from the inside out with a needle.

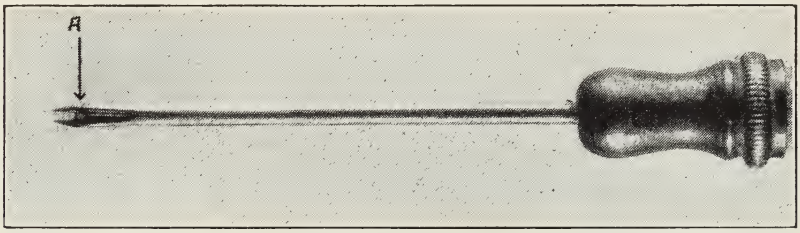

Fig. 25.—A good instrument for vaccinating baby chicks, made by cutting off the end of a large sewing needle through the eye and grinding sharp points on the ends. A tiny drop of vaccine is carried between the two points. It is fastened in a wood or metal handle or a cork. (Greatly enlarged.)

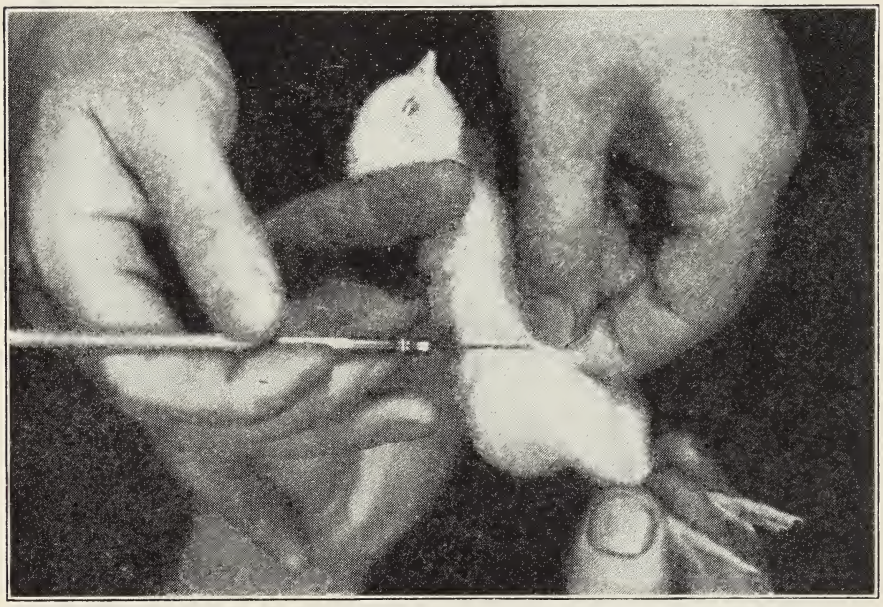

Fig. 26.—Vaccinating a baby chick in the skin of the flank anterior to the thigh.

One or two sewing machine needles attached to a suitable handle, as shown in figure 25, make an excellent instrument. Vaccine carried by both the eye and groove of the needle is quite certain to be deposited in the skin punctures.

If baby chicks are to be vaccinated, the procedures vary from the preceding only in that a single needle is used and the best vaccination site is the loose fold of skin in the flank region, *not the web of the wing*

(fig. 26). In some flocks of baby chicks vaccinated in the latter location, many developed lesions about the head, and heavy loss resulted.

There appears to be little difference between the two methods of administering chicken-virus vaccine either as regards efficiency in immunizing against the disease or effect on the general health of the fowls, except that the stick method is the one to use for baby chicks. The choice

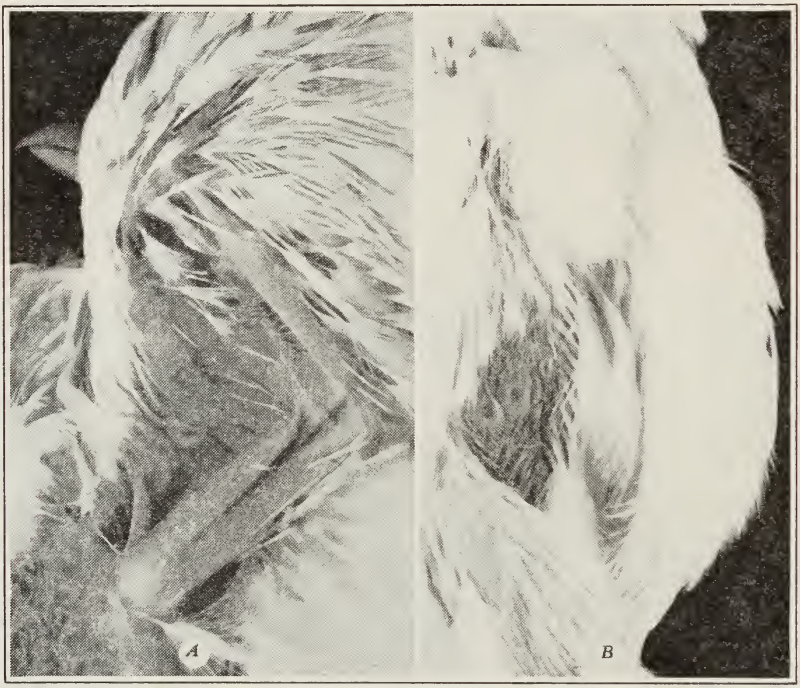

Fig. 27.—Takes from fowl-pox vaccination, by stick method with two needles: *A*, on the web of the wing; *B*, on the breast. *A* also shows accidental infection of a follicle from which a feather was pulled in vaccinating. Photographed on the eighth day after vaccination. (Actual size.)

of method, therefore, is largely a matter of personal preference on the part of the fowl owner or of the operator. Pigeon-virus vaccine, as previously pointed out, must be administered by the feather-follicle method.

Failures to immunize a satisfactory percentage of chickens by vaccination have resulted from attempts of users to stretch the vaccine by vaccinating several chickens with a brush or sticking instrument before again moistening it with vaccine, instead of moistening it between each bird as directed. Such an attempt to economize may prove costly.

Effects of Vaccination with Chicken-Virus Vaccine.—Fowl-pox lesions or takes should develop at the site of vaccination in from 5 to 7 days and remain for 10 to 20 days (figs. 27 and 28). They can be easily

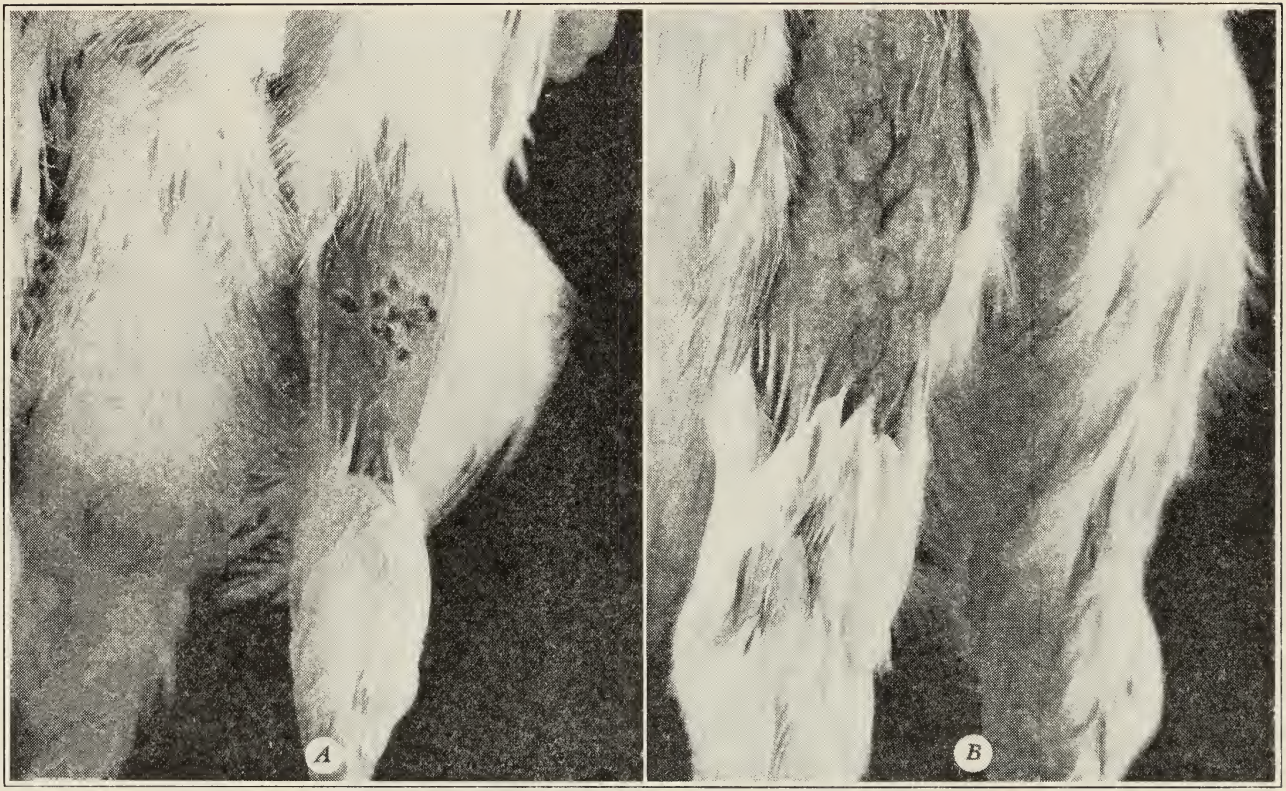

Fig. 28.—Take from vaccination by the feather-follicle or brush method: *A*, with chicken virus; *B*, with pigeon virus. Photographs taken on the eighth day after vaccination. (Actual size.)

detected by sight or feeling as hard nodules by the eighth to tenth day. Takes from virus of chicken origin soon become dry scabs which loosen and fall off. Lesions may develop on the head parts of a few chickens, but these usually disappear quickly and harmlessly. Takes from egg-propagated virus may disappear with slight or no scab formation.

Fowls may become somewhat listless and have a decrease in appetite during the third or fourth week after vaccination. In flocks of young fowls that were in good condition when vaccinated, this reaction may not be seen at all or may last but a few days. The reaction is likely to be less pronounced in fowls vaccinated before they are 3 or 4 months old than in older ones. Healthy adult flocks that are vaccinated when they are molting and laying very few eggs should exhibit little or no effect.

Fowls vaccinated when in poor condition as a result of intestinal parasites, faulty management or feeding methods, or other causes, are apt to have a very severe and prolonged reaction, which leads to continued poor appetite, emaciation, and often death of many birds.

An occasional flock of young birds apparently in good condition when vaccinated suffers a severe vaccination reaction which causes death of many birds and from which recovery is very slow. In some cases, no explanation for the occurrence of a severe reaction under such conditions can be found, but careful investigation is likely to reveal the presence of an intercurrent disease.

Day-old-chick vaccination has been studied in controlled experiments and practiced to a limited extent on farms. In many of the flocks on farms no unfavorable effects were observed; in other instances the results were confused by the appearance of an intercurrent disease. In all controlled experiments, growth of the chicks was definitely retarded by vaccination, although in a number of experimental groups this would not have been detected if the chicks had not been weighed at regular intervals and their weights compared with those of nonvaccinated controls. The harm to the chicks in such instances was not serious nor lasting. The effect of infectious diseases, such as pullorum disease or cecal coccidiosis, however, was much more severe among the vaccinated chicks than among nonvaccinated controls.

The occurrence of colds, infectious laryngotracheitis, or other infectious disease in flocks after vaccination should not be regarded as a direct effect of vaccination or of the introduction on an infectious agent with the fowl-pox vaccine, but rather to infection on the premises, to which they would have been exposed and with which they probably would have become infected whether or not they were vaccinated.

In pullet flocks whose egg yield has reached 10 to 20 per cent or more and in flocks of older hens, egg production can be expected to be reduced

and retarded for a period of 30 to 60 days, and the birds are liable to go through a partial or complete molt. The egg production of pullets that have just started to lay, however, is not so apt to be seriously influenced by vaccination.

Fowls become immunized against fowl pox within 3 weeks after vaccination. The immunity in most cases appears to be sufficiently lasting to protect fowls against the disease for at least a year and usually for life. Vaccination provides no protection against fowl pox for the first 2 or 3 weeks, and during this period will not check the spread of the disease in an infected flock.

Vaccination is indicated:

1. As a routine preventive measure on any farm on which fowl pox has occurred. Pullets (and cockerels to be kept for breeding) should be vaccinated when they are from 1 to 4 months old. If cecal coccidiosis is a regular visitant, it is best to wait until this is past and the flock is fully recovered. When several lots of chicks are raised during a season, each may be vaccinated separately as it reaches a suitable age. Vaccinated and unvaccinated chickens, however, should not be kept in the same or adjoining pens.

2. Routinely on any farm in a congested poultry district whether or not fowl pox has occurred on it. This is advisable to avoid having the disease to combat at a time unfavorable for vaccination. Each age group of chickens can be vaccinated at the most propitious time.

3. For all new stock that is added to a vaccinated or previously infected flock or that is to be placed in houses previously used for vaccinated or infected chickens.

4. For day-old chicks as a routine control measure in broiler plants in which fowl pox is appearing in the chicks before they reach the market age.

Vaccination is not indicated:

1. For a flock affected with another disease.

2. For chickens in poor condition because of faulty management, inadequate diet, intestinal parasites, or other causes. In such a flock, vaccination should be delayed until the cause of the poor condition is removed and the chickens regain good health.

3. For the treatment of infected chickens.

4. For use on farms which are not near other poultry farms and on which fowl pox has never occurred.

5. For performance by persons who are unfamiliar with conditions under which it can be safely and effectively used.

Procedure and Care of Flocks after Vaccination:

1. At least 25 per cent of the flock should be examined for takes on the

eighth to tenth day after vaccination. If a large majority of these do not have definite takes, the entire flock should be revaccinated, or the entire flock examined and those without takes segregated for revaccination.

2. Watch the flock carefully during the second and third weeks after vaccination. If any indication of depression and decreased appetite is seen, endeavor to increase food consumption by tempting the chickens with moist mash, rolled barley soaked in milk, or other appetizing departures from regular feeding practices.

3. When a severe reaction occurs after vaccination, have specimens examined by a competent person to determine if there may not be some complicating condition present which can be corrected.

4. If the reaction after vaccination causes a portion of the birds to become weak and lose weight, separate them from the more vigorous and thereby enhance their chances for recovery.

Some Precautions for Users of Vaccine:

1. If vaccine is used for a portion of a flock, vaccination of remaining susceptible birds should follow soon afterwards.

2. Unvaccinated susceptible chickens should not be kept in quarters previously used for vaccinated or infected chickens.

3. Vaccinated chickens should not be added to a susceptible flock until 2 to 4 months after vaccination.

4. Unvaccinated chickens should not be added to a vaccinated flock.

5. Owners of vaccinated flocks should advise purchasers that the chickens have been vaccinated.

Vaccination with Pigeon-Virus Vaccine.—Pigeon-virus vaccine will produce a take in chickens only when applied by the feather-follicle method. A take consists of swelling of the infected follicles, which disappears in a few days without scab formation.

There is no postvaccination reaction from this vaccine to cause depression, loss of appetite, or decrease in the egg production of laying chickens. Consequently, it has been recommended for emergency vaccination of laying flocks.

The evidence concerning the effectiveness of this procedure is conflicting. Some reports indicate that an exposed laying flock or the still-healthy birds of one already infected can be given protection quickly enough and lastingly enough by pigeon-virus vaccination to ward off the disease and carry the flock until it can be revaccinated safely with the more effective chicken-virus vaccine. Other reports state that resistance to fowl pox develops so slowly from pigeon-virus vaccination that the procedure is of very questionable value. It is generally agreed that pigeon-virus vaccination cannot be depended upon to give protection for longer than about 6 months.

Control of Outbreaks of Fowl Pox.—Sanitary measures that may be used in an infected flock consist in (1) separation of the sick from the healthy and transfer of the latter to freshly cleaned and disinfected quarters; and (2) daily careful inspection of the flock and individual examination at least once a week so that new cases may be detected and removed early. By these measures it may be possible to so retard the progress of the infection that there will never be a large number of chickens affected at a time and the loss from mortality and decreased egg production will be small. These measures are not likely to be effective, however, if a fourth or more of the chickens are found to be infected at the first examination.

Vaccination of birds not yet infected is a certain means of stamping out an outbreak within 3 or 4 weeks. The fewer infected birds there are before vaccination, the more effective is the procedure. For growing birds or nonlaying adults, it can be adopted without hesitation. It should not be adopted for a laying flock, however, without due consideration of the possible adverse effect of vaccination on egg production. To the question as to which will cause the greater loss, the effect of the disease or the effect of vaccination on egg production, no definite answer can be given. If the disease is of a mild form and continues to be, then the disease is perhaps likely to be less costly than vaccination might be, while the reverse is apt to be true if a severe form of the disease is present. Since there are no definite criteria on which to base a recommendation, the choice of procedure must be left to the flock owner.

Vaccination with pigeon virus would be safe, but, as pointed out in the preceding page its effectiveness is somewhat uncertain.

Treatment.—Recovery of infected chickens may be hastened by removing the scabs and canker and applying tincture of iodine liberally with a swab or medicine dropper. Particular pains should be taken to remove all canker from the eyes by careful pressure and massage and from in and around the opening into the larynx with forceps. Treatment should be repeated every 2 or 3 days until definite regression of the lesions is evident.

Medication of the feed and drinking water is valueless.

FOWL TYPHOID

Fowl typhoid is a widely distributed, acute infectious disease of chickens and other species of birds. It also occurs as a localized infection of hens and an acute disease of chicks which is much like pullorum disease. In California, the acute infection of adults is seen more frequently in turkeys than in chickens. This disease should not be confused with the disease of chickens and turkeys known as "paratyphoid."

Cause.—Fowl typhoid is caused by the bacterium *Shigella gallinarum.* The germs are taken into the digestive tract with contaminated food and drink. From here they may enter the blood stream, become generally distributed in the organs, multiply, and cause death; or they may become localized in some organ and not affect the general health. The germs are usually present in the intestines and are discharged from the body with the droppings. The fowl-typhoid organism is closely related to the one which causes pullorum disease, and the effects of the two on chickens may be much the same.

Symptoms.—Sick birds have no symptoms which are typical for the disease. The feathers are ruffled, the wings droop, and the birds become weak. Appetite is always reduced, but thirst may be increased. Diarrhea is a common and often the most prominent symptom. The comb and wattles are frequently pale, but may be congested and dark. Death usually does not occur until after a few days of illness. Outbreaks may follow a prolonged erratic course with few cases appearing at a time.

The lesions in dead birds are no more characteristic than the symptoms of the sick. The changes sometimes found which are suggestive of fowl typhoid are a watery condition of the blood, enlargement and dark red color of the spleen, and enlargement and dark bronze color of the liver.

Infection, when localized in the ovary, produces changes in that organ like those of pullorum disease (see p. 27).

Diagnosis.—Since recognition of the disease is impossible from the appearance of sick and dead birds, a diagnosis can be made only through bacteriological procedures at a laboratory.

Prevention.—Since fowl typhoid is a filth-borne disease, sanitary measures are necessary for its prevention and control. Outbreaks of the acute type of disease seldom occur in flocks that receive proper care and are provided with sanitary surroundings.

In combatting an outbreak, sick birds should be removed, killed, and burned, and the healthy confined to clean, disinfected houses. The litter should be changed frequently and contamination of feed and water with droppings prevented. A disinfectant may be added to the drinking water to destroy any germs with which it may become contaminated (see p. 25). The yards should be thoroughly cleaned and not used again for several months.

When there is localized ovarian infection, the disease may be transmitted to chicks through the eggs. The problem of control is then the same as for pullorum disease and the same measures apply (see pp. 31–36). Fortunately the fowl-typhoid and pullorum-disease organisms are so much alike that the agglutination test for pullorum disease will detect also the carriers of fowl typhoid.

Treatment.—Medication of individuals or through the feed and water cannot be expected to benefit chickens with fowl typhoid. Vaccination with fowl-typhoid or avian mixed bacterin is of uncertain value for either treatment or prevention.

FOWL CHOLERA

Fowl cholera may exist as an acute, rapidly fatal, infectious disease or as a chronic infection which may produce a lingering, debilitating disease or a localized disease process in some organ. All species of domesticated birds are susceptible. In California as a whole, the disease is not of great economic importance; but in certain areas of the state, it is quite troublesome.

Cause.—The disease is caused by a species of bacteria named *Pasteurella avicida.* The organism belongs to a group which infects nearly all species of domesticated animals. The organism which infects one kind of animal, however, is not likely to be infectious for another. Fowl cholera is extremely virulent for small laboratory animals, especially the rabbit. This characteristic is very useful for identifying the organism in the laboratory.

The virulence of the bacillus for chickens may be so low as to be practically harmless or so great that a minute dose will produce rapidly fatal disease. Strains of the organisms of low virulence maintained in a laboratory, are apt to remain in that state; but on farms, through influences of which nothing is known, they may suddenly become highly virulent. Consequently, infected farms may be periodically confronted with extensive and highly fatal outbreaks and in the intervals between enjoy complete freedom from the disease.

Symptoms.—The first indication of an acute outbreak frequently consists in finding birds dead under the roosts or in the nests. As the disease progresses, the virulence becomes less marked and fowls may linger several days before death. The sick fowls are drowsy, their feathers are ruffled, and some may become lame from infection of the joints. Some fowls may survive in a nearly lifeless state for several days, during which they become extremely emaciated. Diarrhea is a constant, although not diagnostic, symptom. The outbreak is likely to terminate as suddenly as it began.

Some of the survivors of an acute outbreak are likely to be healthy carriers of the infection.

In less virulent infections, small but continuous losses may occur over a period of weeks or months. The deaths may take place while the chickens are still in good flesh or after a lingering illness with consequent emaciation.

Fowl cholera also occurs as a localized infection of some organ or part, such as abscesses in various locations, inflammation and swelling of the joints of the legs, infection of the middle ear, edema of the wattles (see p. 75), and infection of the nasal passages. The last two of these are of some economic importance in California.

The lesions in dead chickens are neither constant nor characteristic. The organs of chickens that die suddenly may show very little change. A common and highly suggestive finding is very small, bright-red hemorrhagic spots on the surfaces of internal organs, particularly the gizzard and heart, and on the lining of the body cavity. Another is the presence of numerous small grayish specks or minute areas of necrotic tissue on the surface of the liver. Such lesions, however, are seen in other diseases and are not diagnostic of fowl cholera. The lesions otherwise consist of degenerative changes of the organs like those which may result from various other disease conditions. Yolk material, either in a liquid state or of a cheesy consistency, may be found in the abdominal cavity of laying hens that die from an acute attack of the disease. This condition is discussed further on page 111, in the paragraph on ruptured yolk associated with nonspecific disease of the reproductive organs.

Diagnosis.—A positive diagnosis of fowl cholera can be made only by a bacteriological examination. Neither symptoms nor lesions are sufficiently characteristic. When the presence of the disease is suspected, specimens should be submitted to the nearest laboratory at once.

Prevention.—The prevention and control of fowl cholera must be accomplished by sanitary measures alone (see p. 22–25). The addition of partly grown or adult chickens to farms free from fowl cholera should be particularly avoided since there might be healthy carriers of the infection among them.

When an outbreak occurs, the sick birds should be killed and burned, the healthy confined to cleaned and disinfected houses, and strict sanitation maintained. The yards should be well cleaned and chickens excluded from them for a few months.

Since some of the survivors in an infected flock are likely to be healthy carriers of the infection, measures like those for the prevention of infectious coryza (see p. 58) should be adopted. This would apply either after an acute outbreak or after nasal infection with a strain of the organism of low virulence. The means for preventing fowl cholera when it occurs as edema of the wattles are discussed on page 73.

Treatment.—There is no evidence that drugs of any kind, given in any way, will benefit a sick bird or influence the course of an outbreak through a flock. Neither can any good results be expected from the use of vaccines.

EDEMA OF THE WATTLES

Edema of the wattles, previously mentioned as sometimes accompanying infectious coryza (p. 55) and fowl cholera (p. 74), often occurs independently of those diseases. Chickens of either sex may be affected, but it is of particular economic importance in breeding males. It has been responsible for incapacitating many males after they reached maturity and thereby, in many cases, has seriously interfered with a breeding program.

Cause.—The condition appears to be due to any one of several species of bacteria. One of these, the fowl-cholera organism, has well-established pathogenic properties for chickens; the others are ordinarily harmless. Injection of cultures of the organisms isolated from infected wattles has given irregular results.

Symptoms.—The infection starts as a soft swelling of one or both wattles, which gradually increases in size and becomes more firm. The fluid in the wattle, at first clear and watery, soon thickens and becomes yellowish. It continues to thicken until about the sixth or seventh day, when it has changed to a solid, cheesy mass which can be cleanly removed if the wattle is cut open. The latter then begins to shrivel and becomes reduced to one-third or one-half its original size.

Affected birds become listless, lose appetite, and their weight rapidly decreases. In this condition, if not isolated, they are objects of attack by other males. A high percentage of birds so attacked are killed and many of the others are so badly injured that their usefulness as breeders is destroyed.

The course of the disease from the first symptom to recovery, as a rule, is not more than 3 weeks. A considerably longer time is required, however, for them to regain lost weight and become serviceable breeders.

Prevention.—This trouble can be easily avoided by cropping the wattles of all males in the flock. This can be done at any time after the cockerels are large enough for the first selection of those which are to be retained as breeders. If performed early, the hemorrhage is less and easier to control. The wattles can be cropped by simply cutting them off with scissors or a sharp knife. It is better, however, to use heavy 10-inch compression or Fergerson forceps. These are firmly fixed to the wattle as near the lower jaw as possible and the wattle severed with scissors or knife along the lower side. The forceps are left in place for a few moments after the wattle is severed, and the cut surfaces dusted with antiseptic powder. The use of forceps greatly reduces the amount of hemorrhage; in fact, they are almost a necessity to avoid fatal hemorrhage when the wattles of large birds are cropped. If any cases of edema

of the wattles are present in the flock at the time of cropping, *all instruments should be thoroughly disinfected between each bird.* Failure to do this may result in spread of the infection and heavy loss.

COLIBACILLOSIS

"Colibacillosis" is the term applied to disease conditions of chickens which seem to be of an infectious nature but which yield only the colon bacillus on bacteriological examination. This bacillus is a normal inhabitant of the intestine and usually is harmless. But under certain conditions, not at all understood, it appears to be able to invade the blood stream and tissues and cause disease. Attempts to reproduce the pathological condition with which it was associated by injecting chickens with cultures of the organism have seldom been successful. The presumption that it caused the disease condition with which it was associated is based solely on its presence there.

Colibacillosis is seen principally in young laying chickens and in young chicks. In the former, the disease may resemble fowl cholera so closely that it would be mistaken for that disease if a diagnosis was based on post-mortem findings alone. In such cases, the disease appears suddenly, causes the death of several chickens within a few days, and then as suddenly ceases. For example, in an outbreak in 1938, 17 of a group of 250 vigorous pullets died within 6 days, 12 of them during the first 2 days. The lesions found were fibrinous exudate in the heart sac, congestion of and areas of degeneration in the liver, peritonitis, and ruptured egg yolk. None of the other pens of chickens on the premises were affected at the time and there has been no recurrence. The course of the disease may be more protracted and the losses relatively larger than the case cited.

The disease in chicks has no special identifying characteristics. When present, the losses are above normal but not disastrously high. The organs of dead chicks are apt to show little change beyond some areas of degeneration in the livers of some. Flocks of chicks most likely to be affected are those which are poorly housed, undernourished, or lacking in vigor for some other reason.

There are no special measures for prevention and the control of outbreaks. Good sanitation and proper nourishment and care should be provided.

AVIAN TUBERCULOSIS

Tuberculosis, formerly plentiful in California and still very prevalent in some sections of the country, is rarely seen here at present, and then only in general farm flocks. The reason for the disappearance of tuberculosis from the specialized poultry farms in this state is probably related

to the large percentage of the flocks which are replaced each year with baby chicks. This keeps the average age of the chickens low and eliminates the older birds which would be the most potent carriers and spreaders of the infection. Improvements in sanitation have also undoubtedly contributed much to the reduction of the disease.

Cause.—Tuberculosis is caused by the germ *Mycobacterium tuberculosis.* Filthy, damp, crowded houses and yards are favorable for the perpetuation and spread of the infection but cannot initiate it.

Fig. 29.—Tuberculosis nodules on the liver and the intestines of a fowl. (From Ext. Cir. 8.)

Symptoms.—Affected birds exhibit symptoms of infection only in the advanced stages of the disease. Then they become emaciated, the comb and shanks are pale and shriveled, and some are lame. Losses from this disease do not often occur in birds less than one year old.

The lesions of tuberculosis consist of round yellow spots varying in size from a pinhead to a pea in the liver and spleen, and nodules varying in size from a pea to a small walnut on the intestines or on the membrane which holds the intestines in place (fig. 29). Lesions also occur less frequently in the kidneys, lungs, skin, and bones. For a definite diagnosis the presence of the organism must be demonstrated in the lesions by microscopic examination.

Tuberculosis spreads by fowls' eating substances, such as food and soil, contaminated by the discharges from infected birds. The loss from tuberculosis is not apt to be great at any one time, but it is constant and becomes increasingly severe as time goes on.

Control.—Observing strict sanitary precautions, keeping chickens of different ages in separate pens, and disposing of all fowls when they reach the age of 18 to 24 months should gradually reduce the amount of the disease and eventually eradicate it. For immediate eradication, the entire flock can be sold and the farm restocked with baby chicks. Houses and equipment can be used again shortly after thorough cleaning and disinfection. Yards, however, must not be used for about 3 months if the soil is thoroughly dry and for from 6 months to as much as a year if it is moist.

Another method of controlling tuberculosis is by means of the tuberculin test. This test is made by injecting a small amount of a test substance known as "tuberculin" into the skin of the wattle. If an injected bird has tuberculosis, the wattle will swell. Repeated tests at intervals of about 6 months are necessary to free a flock entirely from tuberculosis by this method. After each test all reacting birds must be removed from the flock and the houses given a thorough cleaning and disinfecting. Because of the expense involved, this test is recommended only for flocks that are valuable as breeders.

LYMPHOMATOSIS

Lymphomatosis belongs to a group termed "neoplastic diseases"; these include all new growths, which are usually referred to as tumors. Because of their insidious nature, the importance of neoplastic diseases is likely to be underestimated. Generally deaths are not great at one time, but the continued loss of individual chickens over the period of a year may amount to a considerable number. Few poultry raisers are fully aware of the extent of mortality in their flocks from this cause.

Neoplastic growths may arise in any tissue of the body, hence there are many types. Lymphomatosis is by far the most important and frequently encountered member of the group in chickens. Some types of lymphomatosis are not universally considered as neoplastic, or tumorous, growths. The general character of the disease resembles a tumor, however, and the two have other characteristics in common.

Tumors not related to lymphomatosis such as those of the connective tissue, called "fibromas" or "fibrosarcomas," occur in widely distributed parts of the body with considerable frequency and do not always impair the function of the organ or tissue involved or the health of the bird. Another such tumor of the intestinal tract, called "adenocarcinoma," is often encountered. Many other types of tumors of relatively little economic importance are also found. An accurate diagnosis of most types of neoplastic growths can be made only by microscopic examination of the tissues.

Lymphomatosis occurs in three types:[18] (1) neural lymphomatosis, commonly known as fowl or range paralysis; (2) visceral lymphomatosis, commonly known as "big-liver disease" or simply "big livers"; (3) ocular lymphomatosis, commonly known as "blindness" or "gray eyes." The three types of lymphomatosis differ only with respect to the organ or tissue which is involved. Neural lymphamotosis indicates that nerves are involved; visceral, that an internal organ or tissue, such as the liver or spleen, is affected; ocular, that tissues of the iris of the eye are invaded.

These conditions are characterized by the infiltration of the tissues with an abnormally large number of cells, the appearance of which is like the lymphocytes of the blood. The effect of this infiltration is, first, merely to displace the tissue cells; but, as the process progresses, the tissue cells may degenerate and be replaced by the foreign cells and the function of the organ or part be impaired or destroyed. The size of the whole or a portion of the invaded organ is always increased. If the process continues long enough, large tumorous masses are produced.

The disease is extremely plentiful and widely distributed. All three types are commonly represented in an affected flock, with one of them predominant. A single type or any combination of the three may be found in one chicken. The relative amount of each in a flock may change from year to year. Poultry raisers are most familiar with types 1 and 3 because of the ease with which they may be recognized. Lymphomatosis is credited with being responsible for nearly half of the mortality among young laying chickens.

Lymphomatosis is often discussed in the literature as a type of leucosis or a part of the avian-leucosis complex. The term "leucosis" includes a group of pathological conditions which are characterized by an enormous increase in the numbers of certain types of blood cells, either in the blood itself or in the tissues. The principal members of the group are termed, according to the type of blood cell that is present, "erythroleucosis," "myeloid leucosis," and "lymphoid leucosis," which includes lymphomatosis. Some investigators find the three conditions constantly associated and, therefore, regard them as different expressions of one disease. Others have found them occurring alone and accordingly are inclined to view each as a disease entity. By some, erythroleucosis and myeloid leucosis are considered to be identical and distinct from lymphomatosis, and still others have expressed doubt that the three types of lymphomatosis are related. Present knowledge of the problem is insufficient to provide an explanation of these variable findings. It may be that the

[18] A fourth type called "osteopetrosis" which affects bones and has been found in a few California chickens is omitted because of its rarity thus far and also because its relationship to the other three types of lymphomatosis has not been fully established.

various types do represent a single disease and that the differences in manifestations are due to the existence of different strains which produce varying responses in chickens. Another possibility is that the association of various types in a flock represents the coincidental occurrence of distinct diseases.

Lymphomatosis is very prevalent among chickens in California. Erythroleucosis and myeloid leucosis, on the other hand, have been seen but rarely and, up to the present, not at all in association with lymphomatosis, and further discussion of them is, therefore, omitted in this bulletin.

Cause.—Although there is a considerable accumulation of evidence, gained both from field observations and experimental results, that lymphomatosis is a transmissible disease, it cannot be said at present that this is an established fact. The many reports of uniformly successful experimental transmission of the disease by some investigators are opposed by the irregular or negative results of similar experiments by others. Furthermore, none of this vast amount of research has succeeded in determining the nature of the causative agent or stimulus of the disease. Bacteria, as a cause, seem to have been definitely eliminated. Some evidence of the presence of a virus has been advanced but this is still inconclusive.

The genetic constitution of chickens has been clearly shown to have an important influence on the occurrence of the disease. The effect of the hereditary factor, however, is believed to be on the resistance or susceptibility of the chickens. In other words, it is not the disease or the causative agent that breeding chickens may pass on to their progeny, but the relative ability of chickens to withstand the disease when they are being kept in a given environment. Chickens which have high resistance to the disease on one farm, however, may not exhibit that quality when they or their offspring are taken to another.

The former belief that intestinal parasitism was a cause of neural lymphomatosis or paralysis has been entirely discredited.

Symptoms of Neural Lymphomatosis.—Neural lymphomatosis may affect nerves in any part of the body, and the symptoms are correspondingly varied. In some cases, a single nerve is involved; in others, two or more. The disease became known as "paralysis" because of the frequent involvement of the sciatic and brachial nerves, which supply, respectively, the legs and wings. The first indications of the affection of these nerves are drooping of a wing or incoördinated movements in walking. The latter is followed by lameness or limping and finally complete inability of the fowl to stand (figs. 30, 31). The paralysis may be of either one or both legs, but one leg is apt to be more severely affected than the

other. The majority of paralyzed birds have a good appetite and, even though unable to stand, retain some ability to move their legs and make vigorous efforts to move about. They assume various peculiar postures,

Fig. 30.—Seven-month-old pullet with paralysis. (From Ext. Cir. 8.)

Fig. 31.—Four-month-old pullet with paralysis. (From Ext. Cir. 8.)

two of which are illustrated in figures 30 and 31. Paralyzed birds lose flesh, the combs become pale and shriveled, the shanks pale, and the plumage rough and dirty.

Another manifestation seen in many nonparalyzed chickens is progressive emaciation, commonly termed "going light" or "fade-out," resulting from involvement of the vagus nerve which supplies the gizzard, intestines, and other vital organs. These symptoms, however, are due just as often or more frequently to other causes, such as intestinal parasitism.

Twisting and jerking of the head and neck are seen when nerves in the neck are affected, and an occasional case of spasmodic coughing has been traceable to involvement of nerves going to the respiratory organs.

Affected nerves are enlarged (fig. 32) and, in many cases, the color is yellowish or grayish. The fine lines, or striations, so clearly visible on the larger normal nerves, are often obliterated. The enlargement is due to infiltration of the nerves with lymphoid cells.

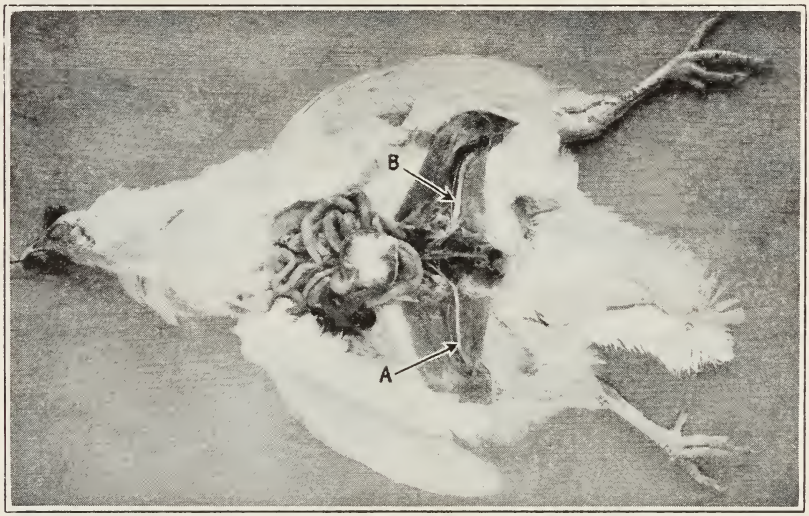

Fig. 32.—Dissection of fowl with paralysis showing (*A*) right sciatic nerve of normal size and (*B*) left sciatic nerve greatly enlarged. (From Ext. Cir. 8.)

The duration of the disease from the onset of visible symptoms to termination in death varies greatly. Paralysis of the legs may progress from slight impairment in gait to complete prostration and death within a few days. On the other hand, prostration may not occur until weeks after lameness is observed. Likewise completely prostrated birds may linger for days or weeks if their appetites are not impaired and food and drink are made easily accessible to them. Paralyzed birds that remain in a good state of nutrition sometimes may regain ability to stand and walk in a normal or nearly normal manner for short periods. Yet rarely, if ever, has permanent recovery been known to occur among birds that have exhibited definite symptoms.

The incidence of neural lymphomatosis is highest in chickens during the first year of life. Numerous cases may appear in an affected flock throughout the first laying year. Extensive outbreaks in flocks as young as 6 to 8 weeks have been reported. As a rule, the disease spreads slowly and only a few chickens are affected at a time. The total number of cases

may vary from a few to as many as 50 per cent of a flock during a period of 4 to 8 months.

Symptoms of Visceral Lymphomatosis.—Visceral lymphomatosis does not have any characteristic symptoms. Affected chickens may become dull and lose flesh, and their combs pale, or they may appear healthy right up to the time of their death.

A common post-mortem finding is an enormously enlarged, pale-colored liver. Along with this, a similar change of the spleen is usually seen and, in many cases, also of the kidneys (fig. 33). The surfaces of the organs may be uniformly pale or mottled. The invading cells may become concentrated in restricted areas to form yellowish or grayish tumors, varying in size from very small to as large as a walnut (fig. 33). Grayish, tumorous enlargement of the ovary and diffuse or tumorous thickenings of the membranes of the abdominal cavity are seen frequently. The heart, lungs, and other organs and tissues may also be involved. The disease may affect the whole or a part of one or more organs at a time, with resultant enlargement of the organ, varying from slight to enormous.

Visceral lymphomatosis has much the same age distribution as the neural type, but its beginning tends to be a little later than the latter and it is more prevalent in chickens more than a year old. Since the affected chickens show no identifying symptoms before death, the actual amount of this type of disease in a flock is usually unknown. The limited data on this point, however, suggest that the incidence of visceral lymphomatosis is likely to be as great as that of neural lymphomatosis.

Symptoms of Ocular Lymphomatosis.—Ocular lymphomatosis begins with a change in the color of the iris of the eye from yellow, yellowish-brown, or red, to gray. As the disease progresses, the pupil becomes constricted and distorted, and loss of eyesight finally results. In some flocks, this type of lymphomatosis is more plentiful than the others.

Diagnosis.—The definite identification of lymphomatosis must be made by microscopic examination of affected tissue. After the presence of the disease in a flock has been established, however, symptoms and lesions are fairly safe criteria for a diagnosis.

Paralysis of the legs or wings, or lameness, can be diagnosed as neural lymphomatosis only if an accompanying definite enlargement of the nerves to the affected part is found. Neural lymphomatosis in other locations does not cause characteristic symptoms. These cases can be recognized only by the demonstration of characteristic gross or microscopic changes in the affected nerves at post-mortem examination. Microscopic examination is necessary for the satisfactory diagnosis of any borderline cases.

Visceral lymphomatosis, as previosuly pointed out, causes no symptoms which are diagnostic aids. The finding, on autopsy, of a greatly enlarged, pale liver, with or without an accompanying similar change of

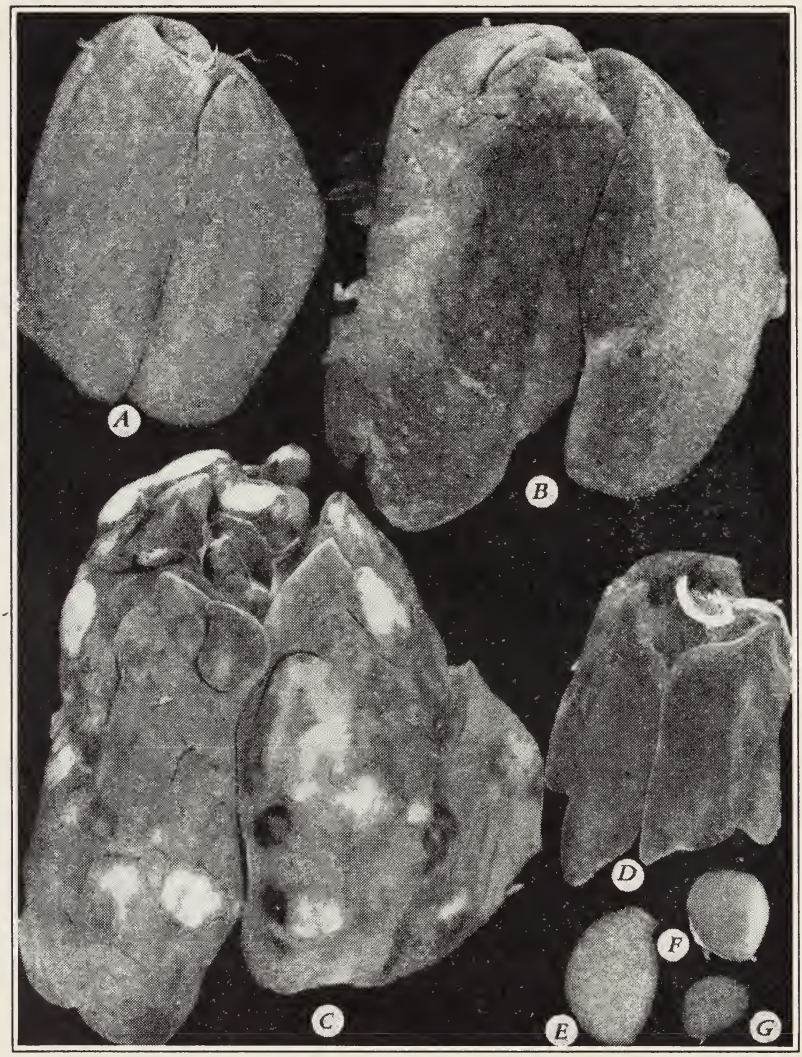

Fig. 33.—Livers and a spleen of chickens with visceral lymphomatosis: *A*, *B*, enlargement of liver from infiltration with lymphoid cells; *C*, liver with tumorous type of the disease; *D*, normal liver shown for comparison; *E*, lymphomatous spleen; *F*, *G*, normal spleens. (All about half natural size.)

the spleen or kidney, is likely to be an indication of this disease. Lesser enlargement and tumorlike lesions of these or other organs require laboratory procedures for identification. But, as previously indicated,

one is justified in regarding these as lymphomatosis when they are found in chickens of a flock in which the disease is known to be prevalent.

Marked grayness of the iris and distortion of the pupil of the eye are reasonably certain to be due to lymphomatosis, especially when they occur in a flock in conjunction with another type of the disease. The diagnosis of a moderate degree of graying of the iris, however, should be confirmed by microscopic examination of the tissue, since there are heritable types of gray iris not associated with disease.

Transmission.—Although, as stated previously, the transmissibility of lymphomatosis is not an established fact, there is both experimental and field evidence which suggests the possibility that the disease may, under certain conditions, spread to healthy chickens by contact with diseased ones or with contaminated litter or other material. No one, however, has demonstrated the manner in which a transmitting agent might leave the body of a sick bird and enter that of a healthy one nor the stage of the disease and age at which this might occur. Some hold to the belief that chickens become affected early in life but that symptoms may not be seen until months later. The idea that, like pullorum disease, lymphomatosis may be transmitted through the egg has little supporting evidence.

Prevention and Control.—While the transmissibility of the disease under farm conditions is not definitely proved nor the manner in which transmission might occur known, it, nevertheless, might be wise to adopt in an affected flock the sanitary measures for combatting an infectious disease. The removal of chickens as soon as they show symptoms and are still in good flesh is desirable because their food value can thereby be salvaged, and possibly the rate of spread may be retarded and number of cases reduced.

At the present time, however, most reliance for prevention and control must be placed on a program of selective breeding, based on the knowledge of the hereditary nature of susceptibility and resistance to the disease. The exact program must be varied in keeping with the nature of the problem.

A breeding or commercial flock that is free from lymphomatosis should have replacements only by stock produced on the farm. A commercial poultryman could produce his own replacements by selecting and mating the best chickens of his flock. An alternative would be to procure the replacement stock from an outside source that is likewise free from the disease. To find such a source, however, would be a difficult and uncertain undertaking.

The proper program for an affected breeding flock is to build up a population of resistant stock by discarding the families in which the

most cases of the disease have occurred. Included in this program should be the autopsy of birds that die so that all cases of lymphomatosis could be detected. A less exact program, and one adaptable also to the type of flocks which supply the bulk of the eggs to commercial hatcheries, consists in using only old hens, and preferably old males as well, for the production of hatching eggs.

Replacements for an affected commercial poultry flock should be resistant stock from flocks in which the selective breeding program has been followed. A commercial flock owner may produce replacements himself by selecting and mating the best of the old birds of his flock and hatching from them.

The inherited resistance of chickens to lymphomatosis cannot be relied upon to protect birds under all circumstances. Under the conditions of experimental transmission experiments and also the natural conditions of a poultry farm, the amount of the disease in rigidly selected resistant lines, after becoming progressively less from year to year, may suddenly take an upturn. The reason for this is not understood. Therefore, the adoption of a selective breeding program to produce a resistant stock and the use of such stock for replacements on poultry farms does not mean that all danger from lymphomatosis has been removed.

Treatment.—There is no method of treatment of chickens with lymphomatosis that is at all effective. Wheat-germ oil, reported a few years ago to be effective for both treatment and prevention of the disease, has been found to have no merit.

COCCIDIOSIS

Coccidiosis exists to some degree on practically all established commercial poultry farms. It is best known to poultrymen as a disease of chicks from 4 to 12 weeks old. It also, however, is a cause of loss among chickens throughout the growing period and well into the first laying year. According to the portion of the digestive tract involved, it is termed "cecal" or "intestinal coccidiosis." This classification has replaced the former, less appropriate one of "acute" and "chronic" coccidiosis.

Cause.—Coccidiosis is caused by protozoan parasites, microscopic in size, which invade and multiply in the walls of the intestine and cause extensive injury to the mucous-membrane lining. Various forms of mismanagement or poor feeding methods, or subjecting chickens to sudden changes in temperature are erroneously believed by some to be primary factors in producing the disease. The only possible relation of such factors to coccidiosis is that they may lower the natural vigor and resistance of the fowls to the effects of the parasites.

There are seven known species of the coccidial parasite which infect chickens. One or more of these are present on practically all poultry

farms. The characteristics which distinguish the species one from the other are the shape and size of certain of their developmental forms, the portion of the intestine affected, the type of injury that they cause to the lining of the intestines, and, in some instances, the symptoms produced.

A species of great economic importance, *Eimeria tenella,* is the cause of the commonly occurring severe, acute infection of the ceca (blind pouches of the intestines) of young chickens, sometimes referred to as "bloody diarrhea."

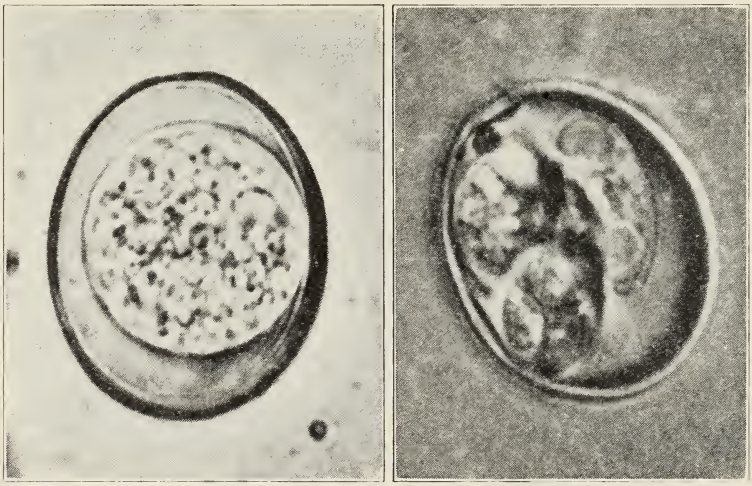

Fig. 34.—Oöcyst of coccidia parasite: left, as it appears in fresh droppings; right, after sporulation. (Greatly enlarged.) (From Ext. Cir. 8.)

The six other species, *Eimeria necatrix, E. maxima, E. acervulina, E. mitis, E. praecox,* and *E. hagani,* infect principally the small intestines. *E. necatrix* causes a very severe and often devastating type of disease. Fortunately it is not so plentiful on California poultry farms as less harmful species. The effect of *E. maxima, E. acervulina,* and *E. mitis,* the intestinal species of greatest economic importance in California, on chickens is less severe, but the total loss is probably greater than from *E. necatrix* because they are more widely distributed. *E. praecox* is of such low pathogenicity that infection with it alone might pass unnoticed. *E. hagani* is a recently isolated species, and little is known about its pathogenicity and distribution.

Chickens acquire infection by eating material which has been contaminated by the droppings of an infected fowl. In the wall of the intestines, the parasite undergoes various developmental changes, multiplies to an enormous degree, and finally produces an egg form or what is termed an oöcyst (figs. 34 and 35). No further development or multiplication can now take place in the fowl, and the oöcysts are discharged with

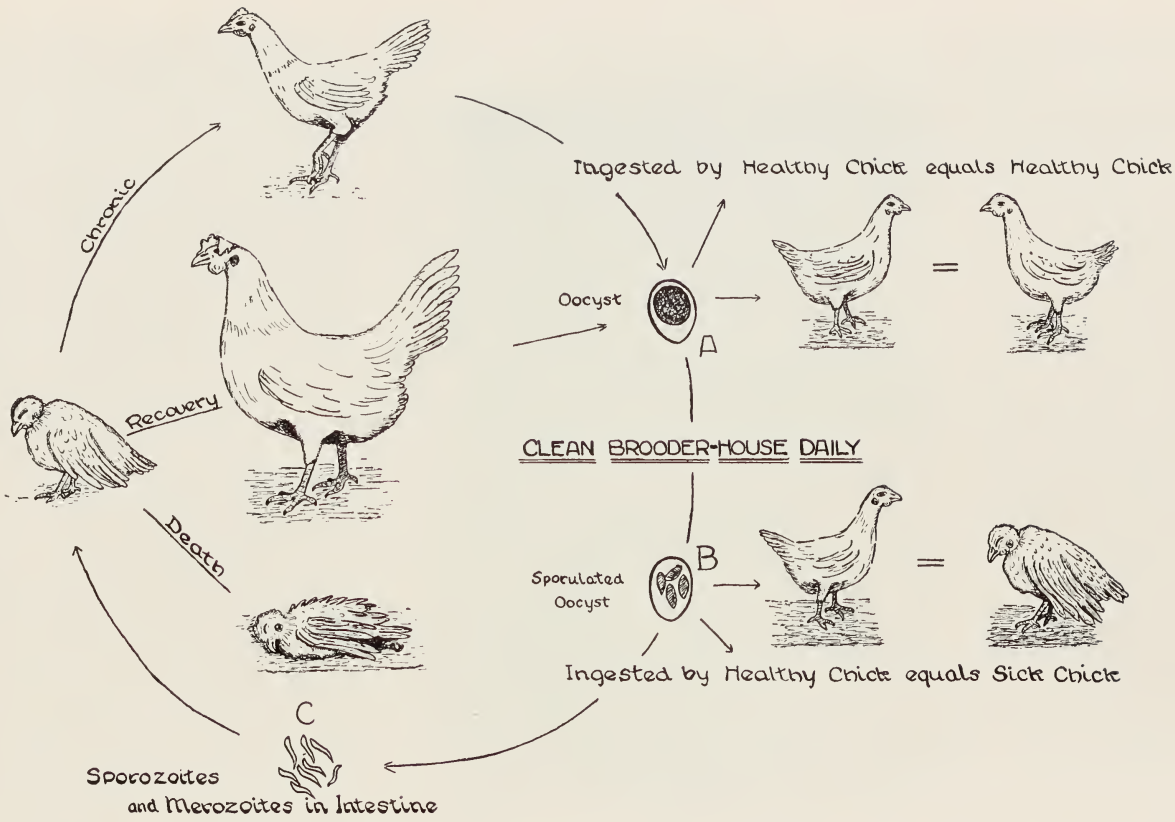

Fig. 35.—Cycle of infection of the coccidial parasite in chickens. *A* represents the noninfective stage in fresh droppings. *B* represents the infective stage in droppings 24 hours or longer after being voided. (From Cir. 300.)

the droppings. The entire process of development and multiplication of the parasite takes place *within* the cells which make up the lining of the intestines. The effect of this invasion is to destroy the cells with resultant sickness and, in many cases, death. In many instances the small blood vessels of the intestinal lining are ruptured and fatal hemorrhage results. The extent of the damage is in direct proportion to the number of oöcysts which are taken in at a time.

The oöcysts in fresh droppings cannot produce disease but, if proper conditions of temperature and moisture are provided, the oöcysts undergo a change known as sporulation (fig. 34). It is the sporulated oöcysts that produce coccidiosis when eaten by a susceptible chicken. Four to 7 days after consumption are required for the parasite to complete its developmental cycle and appear again as oöcysts in the droppings (fig. 35). If a fowl survives an attack for a few days, it will discharge countless numbers of oöcysts and thus expose others. Chickens which survive severe acute infection with certain species of the parasite will cease to discharge oöcysts in a few days, while those infected with certain other species of parasite or with light infection with any species may continue to discharge oöcysts for a considerable period. Coccidial oöcysts are very resistant to disinfectants and exposure to natural conditions on poultry farms. They will survive for months in the soil of poultry yards and in the houses. Consequently, after a farm is once infected, the control of coccidiosis becomes a perpetual problem.

Chickens that survive heavy infection with any species of the parasite or that are exposed to repeated small doses develop a resistance to further infection with that species. The time required and the degree of resistance depend upon the species and the numbers and frequency of intake of the parasites. This resistance may amount to practically complete protection against reinfection or it may be insufficient to prevent reinfection but enough to protect the fowls from any appreciable injury resulting therefrom. In the latter case, chickens may discharge oöcysts over an extended period, during which they are continuous sources of infection of others. Fowls over three years old have been found to be discharging oöcysts with their droppings, although it is certain that they were exposed to infection when they were much younger. *An immunity or resistance to one species of parasite does not protect against infection with any one of the other six.* Thus, for example, chickens that are resistant to *Eimeria tenella* by reason of an attack of the cecal form of coccidiosis are still fully susceptible to any form of intestinal coccidiosis.

Coccidiosis may be distributed from one poultry farm to another by any of the methods of the dissemination of transmissible diseases discussed on page 22. The readiness with which distribution of the parasite

takes place is illustrated by the frequency with which coccidiosis occurs even among chicks that are reared in new houses, with new equipment, on clean soil, and without contact with any other chickens. Infected mature chickens are a likely means of bringing infection to new premises and to young chickens on the same premises.

There is little evidence to support the contention of some that hatching eggs laid by infected mature fowls are likely to be a source of infection of chicks.

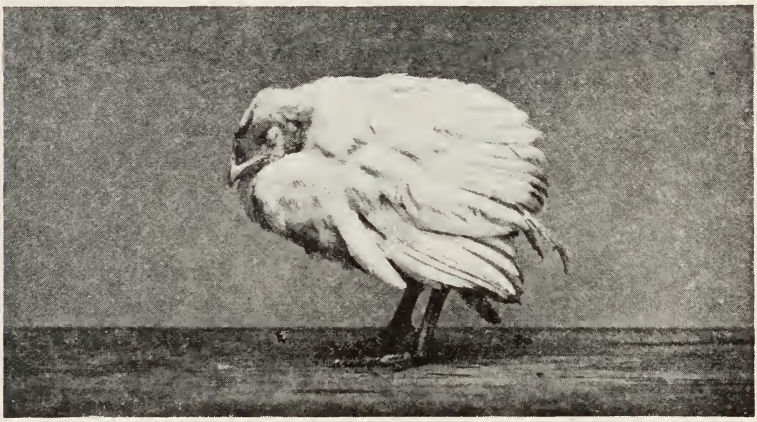

Fig. 36.—Chick with cecal coccidiosis. (From Ext. Cir. 8.)

Symptoms of Cecal Coccidiosis.—As previously stated, cecal coccidiosis is caused only by *Eimeria tenella.* It is usually seen in chickens from 4 to 8 weeks old, although outbreaks among younger or older ones are not uncommon.

In many instances the first indication of the presence of cecal coccidiosis in a flock of chicks is droopiness of a few. The affected birds remain close to the hover, do not eat, and stand with wings drooping, head drawn in, and eyes closed for long periods of time unless disturbed (fig. 36). Droopy birds may be seen for 2 or 3 days before any deaths occur.

The droppings of affected chicks frequently contain blood in amounts varying from that sufficient only to tinge the droppings to enough to give them the appearance of consisting entirely of blood. The bloody droppings usually are seen in the early stages of an outbreak and often are the first indication of disease observed. The sudden deaths in an acute outbreak are due entirely to the loss of blood. If the bloody droppings become mixed with the litter and are overlooked, severely affected chicks may die suddenly without any symptoms having been noticed.

The ceca of chickens which die suddenly at the beginning of an outbreak are usually distended with blood. If the chicks survive a few days,

the ceca contain yellow, cheesy cores (fig. 37). These cores, in chicks that survive, will become gradually reduced in size and be discharged. The light-yellow, cheesy cores sometimes seen in chicks 1 or 2 weeks old are not due to coccidiosis.

Blood in the droppings and bloody or cheesy cores in the ceca of fatal cases are sufficient for a definite diagnosis of cecal coccidiosis. The presence in the droppings of brick-red or salmon-colored, fleshy material

Fig. 37.—Ceca of chick with coccidiosis.
(From Ext. Cir. 8.)

not definitely blood, does not signify coccidiosis, however, because this type of material is frequently seen in the droppings of noninfected chickens.

Deaths in acute outbreaks are most numerous during the first few days, but sometimes continue for 2 or 3 weeks. Many of the survivors may fail to regain normal condition, and never become profitable. The loss from this source is occasionally as great as that from the early mortality.

Coccidiosis is usually present in a flock for some time before it is discovered. It will begin as a very light infection of a few with oöcysts that were present in the brooderhouse when the chicks were put in or that were introduced from without. The infected chicks soon begin to shed oöcysts, which are picked up by others, and so the contamination of the

quarters gradually becomes more concentrated. When proper sanitation is maintained, the contamination may remain so low that none or only a few of the flock will develop symptoms of the disease. On the other hand, the contamination may become so heavy that chicks pick up numbers of oöcysts large enough to cause symptoms and death and an outbreak begins. The initial number of severely affected and dead is determined by the number that have already acquired massive infection, which is naturally a variable quantity. The number of additional fatalities depends largely upon the sanitary measures that are taken to prevent massive infection of other chicks. In any case an outbreak will usually run its course in a week or 10 days. The survivors of infection will, in the meantime, have developed resistance to further infection, and thus continuance of the disease is prevented. Because of this, coccidiosis is termed a "self-limiting" disease.

The loss from cecal coccidiosis, however, is not alone from death. Many survivors of heavy infection will be permanently stunted and not worth keeping. Also moderate infection may occur without symptoms characteristic enough to make the poultryman even suspect that coccidiosis is present. In such cases, the chicks appear unthrifty; some become droopy; some will die; and the dead do not have gross lesions indicative of coccidiosis. Infection of this sort may persist and, in the long run, cause greater loss than more severe infection. Therefore, specimens should always be submitted to a diagnostic laboratory when several of a flock of chicks become unthrifty and droopy from an unknown cause.

Prevention of Cecal Coccidiosis.—Economical and practical sanitary measures offer to poultry raisers an avenue of escape from the enormous losses that result from cecal coccidiosis. The sanitary measures are not intended to eliminate the infection totally but rather to hold it down enough to prevent great damage. The parasites are so widely distributed that chickens are almost certain to pick up the infection sooner or later and, therefore, exposure to mild infection early in life is preferable to complete protection from infection. This enables the chickens to gradually build up resistance against the parasite during the growing period and makes rigid sanitary control unnecessary after they are mature. This statement may suggest that some precaution should be taken against making sanitation too effective. On the contrary, at the present time in poultry districts there are few farms on which a carefully planned and conscientiously executed program of sanitation will do more than keep coccidial infection within safe limits. The essential measures that should be adopted are given in the following paragraphs.

The brooder house and all equipment should be thoroughly cleaned and disinfected before chicks are put in (see p. 15). It should have no

outside runs except sun porches with ½- to 1-inch-mesh wire bottoms. Droppings should not be allowed to accumulate on the supports for the wire, on the wire itself, or underneath. Concrete yards are less desirable than sun porches. When used, they should be cleaned at least once each day or kept covered with litter, which is changed daily. The litter, however, would be objectionable during rainy or windy weather.

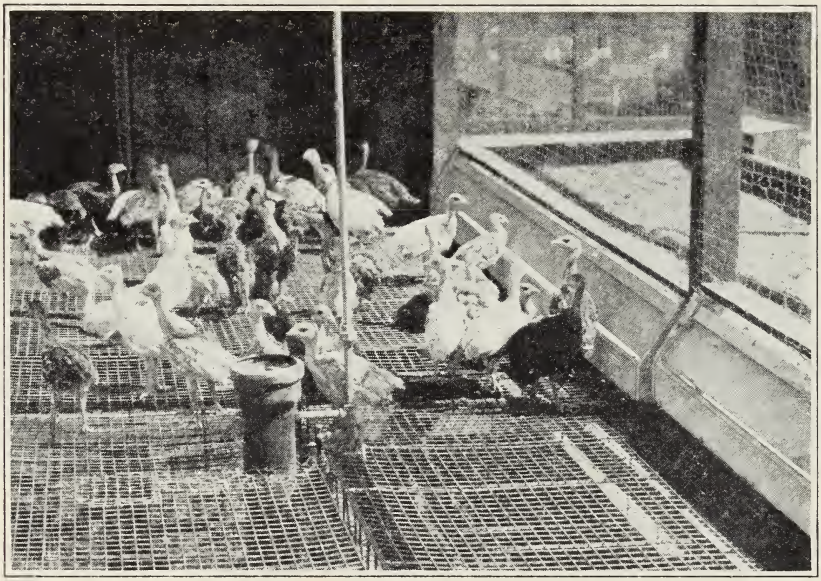

Fig. 38.—An example of sanitary drinking fountain and feed hopper with wire-mesh floor around them to prevent access of the fowls to droppings. (From Ext. Cir. 8; photograph by courtesy of L. Van Es, University of Nebraska.)

The places within houses where coccidia collect in the greatest numbers are around water and feed vessels, under hovers, and beneath roosts. The chickens should be excluded from these sources of infection, after they are 2 or 3 weeks old, by placing the water and feed vessels on frames covered with wire mesh (figs. 3, 6, and 38); installing frames covered with wire mesh beneath the hovers or removing the soiled litter from beneath the hovers and replacing it with fresh once or twice each day; and placing wire netting under the roosts.

On badly contaminated farms, it may be necessary to cover from one half to the entire brooder floor with wire-mesh frames in order to prevent damaging coccidial infection. The wire must be kept free from litter and accumulations of droppings or it will be valueless. The portion of the floor not covered with wire should be swept and fresh litter put in at intervals of not more than a week after the first 2 or 3 weeks.

Feed containers and watering devices should be constructed so that it is impossible for the chicks to deposit droppings in the feed or on top of the containers (figs. 2, 4, 6, 38, and 39).

The houses should be kept dry and not overcrowded.

All of the foregoing sanitary measures should be adhered to as long as the chicks remain in the brooder house.

Colony brooders in plots of growing green feed are reasonably safe, provided there is enough land available so that any given area is not used for more than one lot of chicks each year. The same precautions to

Fig 39.—A feed hopper which prevents chicks from contaminating either the feed or the top of the hopper with their droppings. The chicks are more positively prevented from perching on the metal strip across the top if saw teeth are cut in it. This hopper was especially designed for use in the prevention of coccidiosis. (From Ext. Cir. 8; photograph by courtesy of L. Van Es, University of Nebraska.)

protect the chickens from the places where contamination is likely to be the most concentrated are taken as when brooding is in stationary houses (fig. 40).

The outdoor or colony brooding units with wire-mesh floors are also satisfactory provided the wire and its supports are kept clean.

Considerable study of the value of sulfur in the prevention and control of cecal coccidiosis has been made at several of the agricultural experiment stations. This work has shown that chicks could be protected against artificial infection with sporulated oöcysts by feeding mash containing 5 per cent or more flowers of sulfur for a few days before and a few days after inoculation. Recently the Louisiana Agricultural Experiment Station has reported a method for using sulfur in the prevention of the disease on poultry farms. This is as follows: The chicks are grown in confinement for at least 4 weeks. During this time the sanitary measures previously described should be faithfully carried out. If the chicks are then to be turned out on the ground, mash containing 5 per cent each

of flowers of sulfur and no. 10 charcoal is fed for 2 days before and 5 days after this is done. At the end of this 7-day period, the feeding of an all-mash ration containing 2½ per cent each of flowers of sulfur and of charcoal is begun and continued until the chicks are 12 to 16 weeks of age. The feeding of sulfur in this way is said not to be harmful to the chicks.

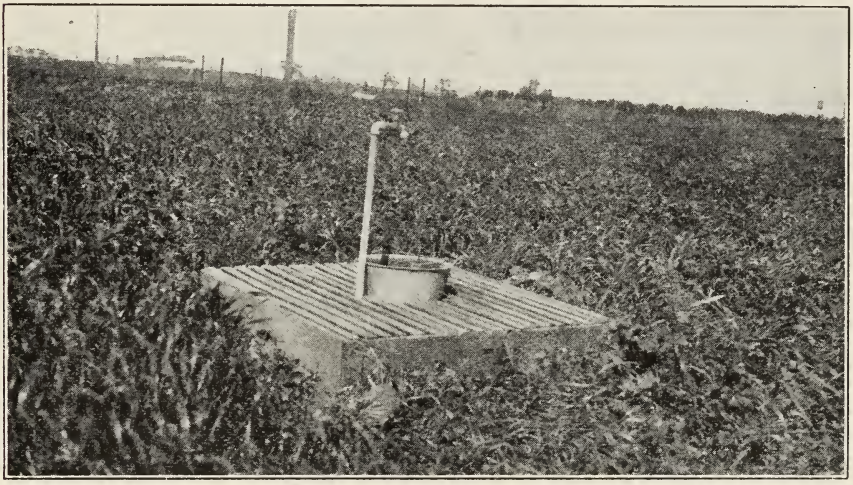

Fig. 40.—An arrangement to prevent moist areas of soil and to protect fowls from droppings around watering places. The soil beneath the slat platform is removed to a depth of 2 feet and replaced by coarse gravel. Any type of water container may be used. The platform should be of such size and shape that all parts of the water container are at least 2 feet from its edge. The use of an arrangement of this sort is of value in the control of chronic coccidiosis and intestinal roundworms. Substitution of wire mesh for the slats would be an improvement. (From Ext. Cir. 8.)

Control of Outbreaks of Cecal Coccidiosis.—When the presence of cecal coccidiosis is discovered, the chicks should be confined in houses equipped to maintain sanitation as recommended for prevention. The litter on the portions of the floor not covered with wire mesh should be changed daily for at least a week, and longer if indications of the disease are still present. The daily change of litter removes most of the oöcysts present in the droppings of infected chicks before they have undergone sporulation, or development to the infective stage, and thereby prevents chicks from picking up massive doses of them. In many cases this relatively simple procedure, when started promptly, is all that is needed to keep fatalities in check until the outbreak is terminated by the self-limiting nature of the disease.

The feeding of mash containing 40 per cent dry skim milk as an adjunct to sanitation is considered beneficial. This has a marked laxative

effect and, therefore, should be fed only a few days. Daily change of the litter is necessary to keep the floor dry. Thirst is increased and so the chicks should be given ample facilities for drinking. If mash and grain are fed, the amount of the latter should be restricted to from one third to one half of the amount of mash consumed. Dried whey may be substituted for dry skim milk; but because of its higher milk-sugar content, the percentage in the mash is 32 instead of 40 per cent. A formula that may be used for home mixing is: dry skim milk, 40 per cent, or dried whey, 32 per cent; wheat bran, 10 per cent; ground yellow corn, 30 per cent; ground barley, 20 per cent with milk or 28 per cent with whey. Forty per cent milk mash is obtainable ready-mixed at many feed dealers.

The way in which the milk-mash benefits the chicks is not definitely known. Some give credit to the milk-sugar content of the milk, some to the nutritional value of the milk, and others to the laxative effect. Those who hold to the latter believe that other laxative foods or drugs would be just as beneficial.

Molasses has been used to some extent as a laxative substitute for milk. There appear to be no experimental data to support this practice. It is usually given as "molasses-bran," a mixture of 15 to 20 parts of molasses with 80 to 85 parts of bran. This is substituted for the mash for half-day periods. Another method of giving molasses is to add 1 cup to each 2 gallons of drinking water.

A method given by the Louisiana Agricultural Experiment Station for using sulfur in the control of outbreaks of cecal coccidiosis is as follows: As soon as symptoms appear feeding of an all-mash ration containing 5 per cent sulfur and 5 per cent no. 10 charcoal is started and continued for the following 7 to 14 days. If the weather is cloudy or if, for any other reason, the chicks do not have at least 30 minutes' exposure to *direct* sunlight each day, the amount of fish oil or other source of vitamin D in the ration should be increased. (One-half pound of fish oil containing 400 A.O.A.C. chick units per gram is said to be ample for 100 pounds of feed.) The medicated mash may be discontinued after 7 days if the outbreak has subsided. Feeding for the 14-day period, however, is said to protect chicks for a longer period and to have no harmful effect if properly done. The recommendations state that sulfur is intended as an aid, not a substitute for sanitary measures and that the latter should be just as rigidly adhered to as if no sulfur were fed.

The results of recent experiments indicate that certain members of the sulfa group of drugs, sulfaguanidine in particular, may be of value in treatment of infected flocks. Thus far, however, the drugs have been used only for treatment of artificially produced cecal coccidiosis and, there-

fore, their effectiveness in outbreaks of the natural disease on farms is not known.

All other attempts to find an effective medicinal treatment for the disease have been fruitless. The drugs and chemicals that have been tried include hydrochloric acid, catechu, mixtures of bichloride of mercury and sulfocarbolates, potassium dichromate, ipecac, and bismuth subnitrate.

Symptoms of Intestinal Coccidiosis.—Intestinal coccidiosis is seen most commonly in chickens between the ages of 4 and 8 months, but it frequently persists throughout the first laying year. Chickens with massive infection with one species of the parasite, *Eimeria necatrix,* have bloody droppings and may die after a brief illness. Exceedingly massive infection with one other species, *E. maxima,* may likewise be rapidly fatal, but in this case characteristic symptoms are lacking. Acute outbreaks or even acute cases, however, are relatively infrequent; instead, the disease is of a lingering, wasting nature, manifested by decreased egg production of laying birds, reduced appetite, roughened, dirty plumage, paleness of the comb and wattles, gradual loss of flesh, and weakness. Death occurs only after several days or even weeks of sickness. Such symptoms would accompany any debilitating disease. The disease may progress so slowly and cause marked symptoms in so few that the presence of a dangerous infectious disease is not suspected. The ultimate loss from retarded development of pullets, reduced egg production, and numbers of worthless culls may, however, be as great as from an acute type of infection or even greater.

The changes found on autopsy of dead birds are confined to the inside of the intestines and vary according to the species of coccidia present. They may consist of any one or any combination of the following: varying degrees of inflammation (reddening) in any portion; thickening of the intestinal lining and the presence of excess amounts of thick, slimy mucus; small, round gray specks (aggregations of coccidia) or red specks (small hemorrhages) in the wall of the intestine, visible from within or without and most numerous in the middle and lower portion; a diffuse thickening and spongy appearance of the lining of the intestines. In severe *Eimeria necatrix* infection, the intestine is ballooned and filled with blood and bloody mucus. Such cases are rarely seen. The gray or red specks in the intestinal walls are fairly typical of coccidial infection, but they and the other changes named can result from other causes; consequently, one is not usually able to identify intestinal coccidiosis without the aid of a microscope. When chickens in a flock of pullets exhibit any of the symptoms or lesions described above, it is advisable to submit specimens to a diagnostic laboratory.

Prevention of Intestinal Coccidiosis.—Sanitation is applied in the prevention of intestinal coccidiosis with the same object as for cecal coccidiosis, that is, to so restrict exposure of the chickens to the parasite that they will not become harmfully infected and will be given an opportunity to develop protective resistance.

The measures to be employed are like those advocated for cecal coccidiosis and need not be repeated in detail. The weekly change of litter should be continued until the chickens are 8 or 10 months old and longer if any clinical cases of the disease are discovered. Emaciated birds should be regularly checked for coccidial infection by a laboratory examination. Confinement to houses and sun porches should be continued throughout the first laying year; after that, danger from the disease is usually past.

The sanitary measures for the prevention of the introduction of infection on a farm should be faithfully followed (see p. 22).

Control of Intestinal Coccidiosis.—When evidence of the presence of a considerable amount of the disease is discovered, the flock should be immediately confined to the house and allowed no outside run except a sun porch. The house should be cleaned daily for about a week. After the first thorough cleaning, a small amount of litter should be used so that it can be easily swept up with a broom. Birds should be excluded from droppings boards by wire netting. The roosts and walks in front of nests and feed hoppers should be swept clean daily. Wire-mesh platforms should be installed around feed hoppers and watering devices and be kept free from litter. Visibly sick chickens should be removed from the main flock so that they may have a better opportunity to recover. Any modification of feeding methods that may increase the appetite should be tried. Sometimes a moist mash, frequent feedings of small amounts of fresh, tender greens, or a daily feeding of rolled barley soaked in milk will accomplish this. If the flock continues to show evidence of the disease after a week, the daily change of litter should be continued a little longer. When this is no longer necessary, a weekly schedule of litter change should be adopted. Milk mash, drugs, and chemicals have not been found of value as an adjunct to sanitation in the control of intestinal coccidiosis.

MYCOSIS OF THE CROP

Mycosis of the crop is a common cause of losses in turkeys from 2 weeks to 5 months old, but is of rare occurrence and of little economic importance in chickens. The only reason for including it in this bulletin is that the disease is said to be the cause of unthrifty condition and greater than normal mortality in numerous flocks of chicks. The field diagnosis of mycosis in such cases can seldom be confirmed by laboratory examina-

tion. The condition called "gizzard ulcers" or "erosions" is commonly but mistakenly considered to be due to mycosis.

Cause.—The disease is caused by a fungus called *Monilia albicans,* and usually occurs in company with some other debilitating condition. The organism, therefore, appears to have low virulence for chickens and is not likely to cause any disturbance in a well-nourished, vigorous flock. The exact source of infection in a flock usually cannot be determined.

Symptoms.—Affected chicks do not show any specific symptoms. They become listless, eat little, lose flesh, and the feathers are dirty and rough. There is a small daily loss from death.

The lesions found at autopsy are usually confined to the crop. The mucous membrane is thickened, rough, and dull, and may be covered with a dirty white, false membrane. The organism can be demonstrated by microscopic examination of scrapings from the crop lining. A positive diagnosis cannot be made in any other way.

Prevention.—Mycosis is unlikely to occur in any flock of chicks that is raised in reasonably sanitary surroundings and fed a proper diet.

When the disease has been diagnosed, the chicks should be confined in clean, disinfected houses. Daily cleaning of the floors, feed hoppers, and water vessels should be practiced for at least a week and longer if conditions warrant. The smallest, poorest birds should be killed and burned. The diet should be carefully investigated and any deficiencies found, corrected.

The substitution of a 1–2,000 copper sulfate (bluestone) solution for all drinking water for several days is an aid in control. A solution of this strength is not poisonous but is sufficiently distasteful that chicks will not drink it if other water is available. It should be put in crockery or wood containers. A convenient method of making it is as follows:

For the stock solution dissolve 1 pound of copper sulfate in 1 gallon of soft water or of hard water to which 1 teaspoon of hydrochloric acid or 1 cup of vinegar has been added. Heat may be necessary to completely dissolve the copper sulfate. Store in a glass jar or a crock. To make a 1–2,000 solution, add 1 tablespoon of the stock solution to each gallon of water.

Use the solution in place of drinking water for at least a week after improvement of the flock is noted.

INFECTION OF CHICKENS WITH HUMAN AND EQUINE VARIETIES OF SLEEPING SICKNESS[19]

It has been found recently by investigations conducted at the Hooper Foundation that the viruses of western equine encephalomyelitis (a variety of sleeping sickness of horse and man) and of St. Louis en-

[19] Contributed by Wm. D. Hammon of the George Williams Hooper Foundation for Medical Research, University of California.

cephalitis (a variety of sleeping sickness of man) are common infections of chickens as well as other domestic animals, in certain of the valley areas of California and the Yakima Valley of Washington. To a lesser extent, other birds are affected. In chickens and most other animals, the infection usually takes place without visible or recognizable symptoms, but it can readily be detected by blood tests or tests of the brain and spleen. The virus of the eastern type of equine encephalomyelitis, however, may produce visible illness and even death in some chickens. There is apparently little or no danger that these diseases may be transmitted to man or horses through contact with infected chickens or to man through consumption of them when cooked; but that they serve to infect mosquitoes appears probable. From the high rates of infection in chickens, as demonstrated by blood tests, and the rare infection of man, it appears probable that the insect which is responsible for transmission prefers the blood of lower animals to that of man, in most instances. We have much more to learn of these infections in relation to lower animals and man, but in certain areas where environment is suitable and the insect vector and virus are present, it has been established that infections occur in many domestic animals.

NUTRITIONAL DISEASES

The diseases discussed in this section are those related to known dietary deficiencies which occur on poultry farms.[20] Those that are seen only in chickens on experimental diets are omitted. The investigations of nutritional diseases thus far have been concerned principally with deficiencies in the diet, leaving the field of the pathological effect of excess amounts of food constituents largely unexplored.

VITAMIN-A DEFICIENCY

Disease results when the intake of vitamin A by the fowl becomes less than is necessary to maintain health. In its early stages or in case the deficiency is only partial, the manifestations are indistinguishable from those of coryza, or colds. This symptom is so constant that the disease became known as "nutritional roup." If the ration provides very little or none of the vitamin or if a partially deficient ration is fed over a long period, very characteristic symptoms and lesions develop.

Symptoms.—The symptoms are decrease in appetite; weakness and emaciation; droopiness (fig. 41); reddened and watery eyes which may be followed by the formation of an adherent white film over the third

[20] For a more complete discussion of dietary deficiencies, the reader is referred to: Almquist, H. J., W. E. Newlon, and T. H. Jukes. Feeding chickens. California Agr. Ext. Cir. 108:1–41. Revised 1940.

eyelid and a mass of white cheesy material within the eyelids (fig. 44); the formation of yellowish-white, round, cheesy, pustulelike patches about the size of a pinhead in the mouth and throat (fig. 42); and occasionally the formation of masses of white, cheesy material in the cleft or elsewhere in the mouth.

Post-mortem examination of birds usually shows the kidneys to be very pale and marked with a network of very fine white lines (fig. 43).

Fig. 41.—Typical appearance of fowls with vitamin-A deficiency. (From Ext. Cir. 8.)

Occasionally a deposit of a white material is also found on the surface of the liver, on the membrane around the heart, or elsewhere on the surface of the organs.

Prevention and Control.—The common sources of vitamin A for poultry are any kind of fresh greens, dried greens such as alfalfa meal, yellow corn, and fish oils. The disease will not occur in chickens whose diet contains an adequate amount of vitamin-A-carrying ingredients of good quality. When fresh greens are fed and there is an abundant year-round supply, an adequate amount of vitamin A is assured. Alfalfa meal and fish oils are less certain sources because the vitamin A is so easily destroyed by exposure to air, particularly in warm temperatures. Thus a large part of the vitamin A in alfalfa may be lost by improper curing, and the vitamin A in fish oil and alfalfa meal tends to diminish after these ingredients are mixed in the mash. Consequently only fish oils and

Fig. 42.—An advanced case of vitamin-A deficiency, showing the pharynx and esophagus studded with pustules. (From Ext. Cir. 8.)

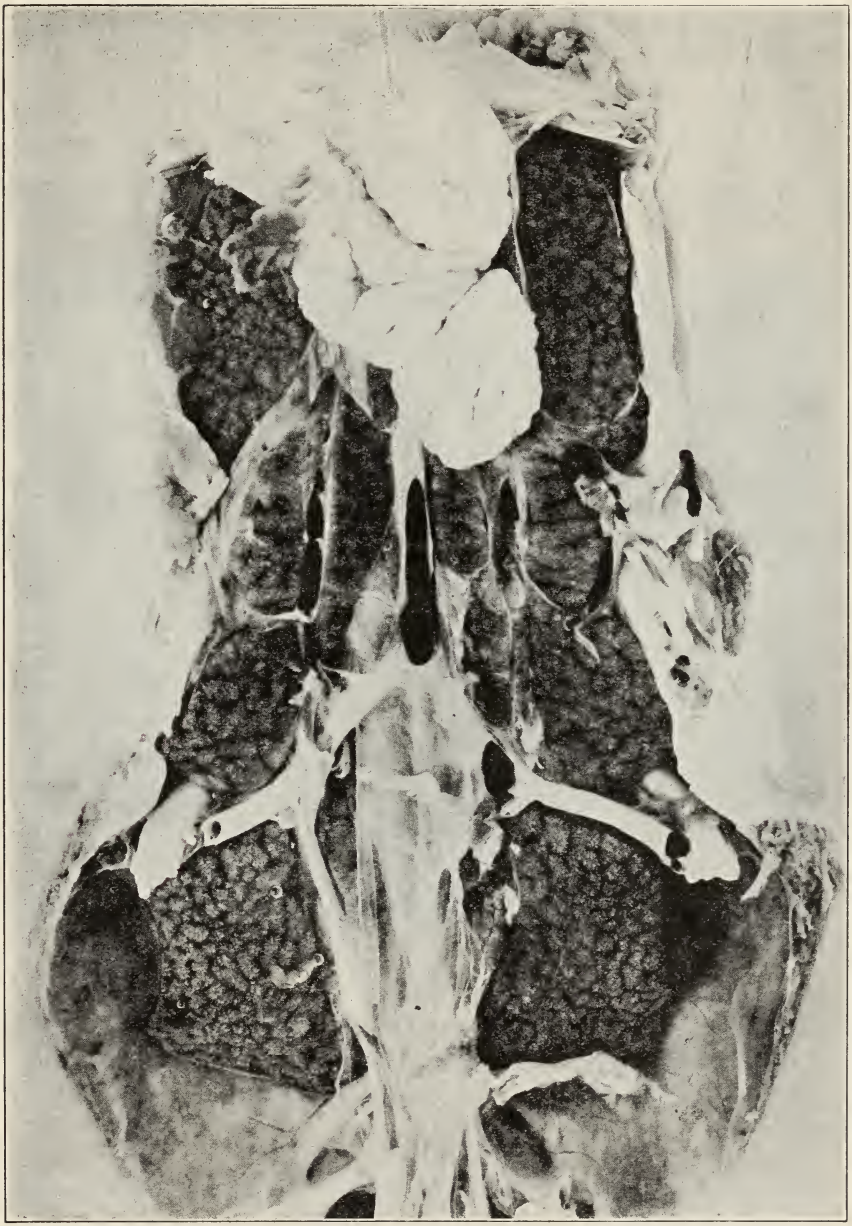

Fig. 43.—Kidneys of a fowl with vitamin-A deficiency. (From Ext. Cir. 8.)

alfalfa having a guaranteed minimum vitamin-A content should be used, and these should be stored so that they are protected as well as possible from exposure to air and warm temperatures and added to mash in relatively small batches.

Flocks or fowls suffering from vitamin-A deficiency recover very quickly when their intake of material supplying the vitamin is increased. It is better to feed fresh greens than to add alfalfa meal or cod-liver oil

Fig. 44.—Cheesy material in the eye of a fowl with vitamin-A deficiency. (From Ext. Cir. 8.)

to the mash, because the appetite of the chickens is impaired and they will eat fresh greens more readily than mash. Very sick birds will respond rapidly to individual dosing with 15 to 30 drops of a good fish oil daily.

VITAMIN-D DEFICIENCY (RICKETS)

Rickets is a disease of the bones of growing chickens caused by a deficiency of vitamin D in the diet. This vitamin can be replaced by ultraviolet light rays. These rays are found in sunlight and can be produced artificially by mercury-vapor or carbon-arc lamps. The effect of these rays is to convert substances in the skin to vitamin D. When this vitamin is absent, calcium and phosphorus are not adequately deposited in the bones.

The symptoms of rickets in young birds are poor growth, weakness of the legs (fig. 45), a stiff-legged gait, and swelling of the joints. "Beading" of the ends of the ribs is a particularly characteristic lesion. Laying hens without adequate vitamin D will continue to lay eggs, but since they cannot utilize the calcium in their diet, will take calcium from their bones in order to make eggshells. This is often the cause of crooked breast bones in good layers.

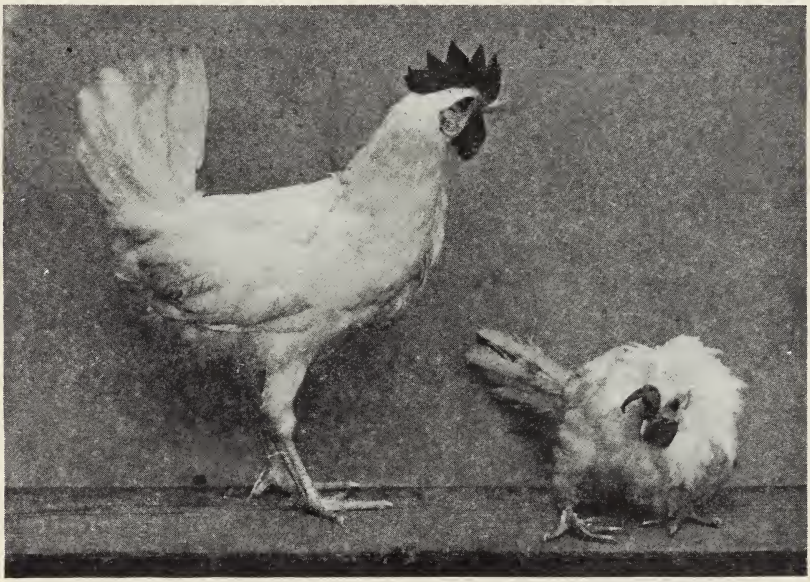

Fig. 45.—These birds were raised together in a well-lighted house and fed the same ration. The one on the left was placed in a box outside in the sunshine for about an hour each day. The one on the right received sunlight only through a glass window. This is a pronounced case of rickets. (From Ext. Cir. 8.)

If chickens have abundant exposure to direct sunlight, no provision for vitamin D in the diet is required. Sunlight coming through a glass window is valueless, however, because ordinary window glass filters out the ultraviolet rays. Young chickens that are confined to the houses a considerable portion of the time because of cool or rainy weather and whether confined or not, during prolonged periods of cloudiness or fogginess, need a vitamin-D supplement in the ration. It is best to supply vitamin D routinely in the diet of chicks for at least the first 2 months of their life.

Fish oils are the best sources of vitamin D for chickens. They are now marketed in containers which give the vitamin-D potency of the oil on the label in terms of units of the vitamin per gram of oil. Directions for use are given on the label and should be followed. When the oil is used

to control rickets already present, a considerably larger amount should be used than is necessary for healthy chicks, until definite improvement of the chicks is seen.

Synthetic vitamin-D-bearing preparations of known potency may be substituted if fish oil is unobtainable.

RIBOFLAVIN (VITAMIN G) DEFICIENCY

Riboflavin is one factor of what is known as the vitamin-G complex. The term "vitamin G," however, is now commonly used with reference to this factor alone. Riboflavin is a yellow pigment that is present in several

Fig. 46.—A chick afflicted with curled-toe paralysis caused by an inadequate supply of riboflavin in the diet. Note the typical posture and the inwardly curled toes. (From Ext. Cir. 108.)

natural foodstuffs. The requirement of chickens for this vitamin is very high. A deficiency of it may cause a characteristic condition known as "curled-toe paralysis" (fig. 46), and also slow growth and poor utilization of food. A deficiency in the diet of breeders causes poor hatchability of eggs.

The prevention and control of riboflavin deficiency naturally consists in feeding an adequate amount. Good natural sources are fresh and dried greens, dried milk, and dried whey. Cereals supply some of the vitamin but are considered poor sources. Pure, undiluted dried yeast is an excellent source but is too expensive. Its use for a limited period to bring a badly deficient flock back to normal may, however, be advisable. Undiluted liver meal is another good source. The requirement of chickens for riboflavin is so high that it may be impracticable to supply the entire need from one source; consequently, it is customary to use both alfalfa meal and dried milk or whey in poultry rations.

PANTOTHENIC ACID (FILTRATE FACTOR OR ANTIDERMATITIS VITAMIN) DEFICIENCY

Pantothenic acid is another factor of the vitamin-G complex which is required by chicks. Symptoms of a deficiency of this vitamin are incrustations at the corners of the mouth; thickening of the edges of the eyelids, which tend to stick together; ragged, brittle feathers; and retarded growth (fig. 47).

Fig. 47.—A chick suffering from dermatitis caused by a lack of pantothenic acid (the filtrate factor, or antidermatitis vitamin) in the diet. Note the incrustation at the corners of the mouth and tightly stuck eyelids. (From Ext. Cir. 108.)

Disease from a deficiency of this vitamin can be avoided and corrected by supplying an adequate amount of it in the ration. The richest natural sources of pantothenic acid among ordinary poultry foodstuffs are cereal grains and wheat bran; consequently, all chick rations should contain as much of these ingredients as practicable. Cane molasses is another good source which may be regularly incorporated in chick rations as a preventive or added to the ration when symptoms of a deficiency are seen in a flock.

Symptoms of deficiency are seen in some flocks which are fed a diet which should supply an adequate amount of the vitamin. These occurrences are sometimes difficult to explain but may be related to the diet of the parents of the chicks or to feeding methods. The usual diets of breeding chickens are apparently never sufficiently lacking in pantothenic acid to affect health or hatchability of eggs, but, if they provide

only a small amount of the vitamin, the chicks will have no reserve store at hatching. Such chicks may not take in enough of the vitamin during their early life to ward off a deficiency even though their diet would be adequate under ordinary circumstances. Similarly, when the diet is mash and grain which contains enough of the vitamin when the two are consumed in proper portions, some chicks may eat mash almost exclusively and thus not get enough of the pantothenic-acid-bearing cereals. In such cases, there would be a few cases of pantothenic acid deficiency in an otherwise healthy flock.

GIZZARD EROSIONS

Gizzard erosions, also erroneously called "gizzard ulcers," are seen in many flocks of young chicks. The erosions consist of patches of the gizzard lining which become discolored and roughened, and finally disappear.

The condition results from a deficiency of a necessary dietary ingredient termed the "gizzard factor," which in some way reduces the production of bile. It is not caused by the fungus infection of the crop and gizzard known as "mycosis" (p. 98).

Gizzard erosions do not appear to retard growth nor to otherwise affect the health of the chicks. They tend to disappear as the age of the chicks increases, perhaps because the need for the gizzard factor becomes less. When found in undersized and unthrifty chicks, as they frequently are, the gizzard erosions must not be regarded as the cause of the poor condition of the birds. The conditions responsible for the sickness of the chicks, however, may indirectly have increased the severity of the gizzard erosions.

The gizzard factor is present in a number of poultry foodstuffs, including fresh and dried greens, wheat bran, rice bran, hempseed meal, and soybean meal. Kale is a better source than alfalfa. The factor can also be supplied by substituting a liquid milk product, that is, skim milk, buttermilk, or whey, for the drinking water. Dried milk products, however, are ineffective. Gizzard erosions will usually be prevented by a ration containing 5 per cent of a good grade of alfalfa meal and 15 per cent of wheat bran. The feeding of granite grit is also an aid in preventing gizzard erosions. When the trouble is known to be present in a flock, it may be advisable to enrich the diet with the protective foodstuffs.

PEROSIS (SLIPPED TENDON)

Perosis, or slipped tendon, is a disease seen principally in young chickens in poultry establishments, such as broiler plants, in which the birds are raised in batteries or pens completely floored with wire mesh. Chickens raised by ordinary methods are seldom affected.

The condition, a permanent deformity, starts as a swelling around one or both hock joints. In a few days, the shank becomes turned outward and bent, and later the tendon may slip from its normal position over the back of the hock (fig. 48).

Fig. 48.—An example of slipped tendon. The tendon of the bird's right leg (on the left in the photograph) has slipped outwards from the normal position, which bends and cripples the leg. The left leg is not affected. (From Ext. Cir. 108.)

Affected chickens show no other abnormality and if able to get enough food remain in fairly good flesh.

The factors which cause it are varied and the severity or prevalence of the disease in a flock is proportional to the number of these factors which operate together. Dietary factors are a deficiency of manganese, a deficiency of choline, too much phosphorus, and perhaps, also, too much calcium. Another factor is hereditary; this causes heavy breeds to be more susceptible than Leghorns. A fifth known factor is confinement on wire floors, but this is not so potent as the absence of dietary factors. A deficiency of manganese in the diet of breeders results in deformity of chick embryos.

Perosis can usually be prevented by avoiding the use of too much bone meal and of meat and bone scraps which contain large amounts of ground bone, and also by the addition of ½ pound of dry manganese sulfate to

each ton of mash. The addition of the manganese, however, will not compensate for errors with respect to other components of the mash. Diets ordinarily used are not likely to be deficient in choline.

NONSPECIFIC DISEASES

This section is included for the purpose of calling attention to the importance of the large group of varied pathological conditions which, as far as is known at present, are unrelated to any infection or parasitism or to nutritional and genetic factors. There is no definite knowledge concerning their cause or of measures for their prevention.

No one of these conditions by itself is likely to cause a large number of deaths in a flock, but the data which have been collected concerning them indicate that together they are probably responsible for nearly half of the mortality and for a fourth or more of the culls among birds of laying age on the average poultry farm.

Considerable information concerning the relative frequency of the different types of nonspecific disease conditions, their distribution in the body, and the age distribution has been obtained by the systematic autopsy of the dead and culls on commercial poultry farms and in the flock of the Division of Poultry Husbandry of the University of California. The data show that the anatomical systems most frequently involved were, first, the reproductive organs, second, the digestive organs (including the liver) and, third, the kidneys. Taken together, these comprise more than 80 per cent of nonspecific diseases. Disturbances of the digestive system were the most prevalent in hens under a year old, those of the reproductive organs thereafter. Kidney involvement had no marked age preference. Nonspecific disease was less prevalent among hens under a year old, that is, during the first half of the first laying year, than in older birds. Stated more specifically, in the flocks studied, approximately one third to one half of the mortality among 5- to 12-month-old birds and approximately three fourths of the mortality among older birds were due to nonspecific disease.

The most frequently occurring abnormalities of the digestive organs were enteritis (inflammation of the intestines), impaction of the crop or gizzard, and degeneration (and, in many cases, rupture) of the liver. The intestinal involvement produced many culls; liver degeneration was usually fatal.

The principal changes involving the reproductive organs were ruptured yolk, with resultant peritonitis; accumulations of all types of egg material, including soft-shelled and fully formed eggs, in the abdominal cavity; and impaction of the oviduct with masses of solidified egg material. Many of these cases were outwardly healthy and were culled because

of unsatisfactory production. The accumulation of eggs and egg material in the abdominal cavity was apparently a result of reverse peristalsis, or backward movement of the oviduct, and the number of such cases was surprisingly large.

The condition termed ruptured yolk, in the preceding paragraph, is so prevalent in some flocks that it has been studied experimentally. It is seen most frequently in pullets during periods of heavy egg production. This suggested that it might be the result of feeding a laying ration that was too stimulating, but this idea was not substantiated by experimental results. In some studies that have been reported, the fowl-cholera or the fowl-typhoid organism was isolated from about half of a large number of cases that were examined, the others being bacteriologically negative. Attempts to reproduce the condition, however, by injecting or feeding laying chickens with bacteria isolated from the ruptured yolk cases resulted in failure. In other investigations, no association between ruptured yolk and bacterial infection was found. These results indicate that the occurrence of ruptured yolk is not dependent upon bacterial infection. Selective breeding studies recently reported indicate that ruptured yolk tends to be more prevalent in some families and consequently that there may be a genetic basis for the occurrence of the trouble.

Death from ruptured yolk often occurs after a very brief illness and may take place so suddenly that no evidence of sickness is observed. Birds may be found dead on the roosts or in nests. The symptoms are droopiness and discoloration of the comb. The constant finding on autopsy is yolk material in the abdominal cavity. This may look like a normal yolk or be of a cheesy consistency. The membranes in the abdominal cavity may be thickened. The abdominal organs, particularly the liver, may be degenerated. Losses from ruptured yolk have sometimes decreased when the diet has been changed so as to reduce egg production. This has been accomplished by diluting the mash in use with an equal quantity of bran or by giving two or three feedings daily of bran moistened with hot water. When the latter procedure is used, access to other feed should be prevented until the bran mash is eaten. Since fowl cholera or fowl typhoid may also be present, specimens should be submitted to a laboratory at once and the sanitary measures applicable to these diseases carried out until the absence of these infections is demonstrated by the bacteriological examination. Owners of breeding flocks which suffer losses from ruptured yolks might obtain some relief by determining whether the trouble is most prevalent in certain families and eliminating those worst affected.

In the birds which died from involvement of the urinary system, the affected kidneys were pale and swollen and, in many cases, engorged

with urates like those illustrated in figure 43. Some of the birds with this condition died quickly and others lingered until they became emaciated and were culled.

These nonspecific diseases constitute a serious cause of death and culls among laying chickens. Until some knowledge is obtained of the causative factors and remedial measures, poultry raisers can be offered no method of prevention of a considerable portion of the present high mortality in their flocks. This emphasizes the importance of making full use of the sanitary measures and more specific procedures for the prevention of infectious and parasitic diseases.

CANNIBALISM

Losses from cannibalism in chicks and young laying chickens are of real economic importance. In some flocks, it is the greatest single cause of death. Whether it is a vicious habit or is related to a dietary deficiency is a much discussed but still unsettled question.

In chicks and growing birds, it usually takes the form of toe, wing, and tail picking. In many flocks of young laying birds and sometimes in older ones, vent picking becomes prevalent. This is often referred to as "prolapse of the oviduct" or simply "prolapse." A prolapse undoubtedly does occur in many cases but would not be fatal if the victims were not picked by the others. In many cases, however, the picking probably starts at a slight injury or hemorrhage of the vent or even at the exposed, reddened mucous membrane after the passage of an egg. The picking in many cases is continued until the victim is completely eviscerated. Vent picking, or "pick-outs" as this is commonly called, is the most damaging type of cannibalism. Feather pulling becomes prevalent in many flocks of partly grown birds and layers. This, in itself, is damaging principally in causing disfigurement of the birds, but the slight bleeding that may follow pulling of the feathers often leads to fatal picking.

The tendency to toe, wing, and tail picking is greatest when chickens are confined in crowded quarters and particularly if the floors are wire mesh. Diets with a low protein level or deficient in salt (sodium chloride) are also said to favor development of the vice.

Vent picking is less closely related to management or diet, but is likely to be more prevalent among closely confined chickens.

There is considerable experimental evidence to indicate that feather picking is influenced by the amount of fiber in the diet; that is, the lower the fiber content of the mash, the greater the tendency to feather picking. The fiber can be supplied by ground oats, ground barley, or wheat bran. The benefit of the fiber is said to be lost, however, if given in pellets instead of in mash. The physical characteristics of the feed would seem

of more significance, therefore, than its fiber content. This suggests further that the cannibalistic tendency is less when chickens are forced to spend a considerable portion of their time in eating. The time element would be much greater with a mash and grain system of feeding than when the food is supplied as pellets.

Some of the many measures that have been proposed for the prevention of cannibalism are as follows:

Avoid raising chickens in large flocks, especially if they are confined in the houses and on wire-mesh floors.

Darken the windows of brooder houses and illuminate with red electric light globes or color the window glass red. This measure, while effective, may necessitate closing the openings to the outside so tightly that it is difficult to provide adequate ventilation.

Add from 0.5 to 2.0 per cent of salt, for 4 or 5 days, to the mash that is being fed. This is said to stop cannibalism within 1 or 2 days. Another "salt cure" that has been recommended consists of the addition of 1 tablespoon of salt to each gallon of drinking water for half of 2 successive days at intervals of 3 days.

Watch the flock carefully so that picked chickens and also the more aggressive pickers can be detected and removed before too much damage is done. Keep the injured chicks segregated until the wound is healed or smear the picked area liberally with pine tar or an antipick ointment and return them to the flock.

An antipick ointment may be obtained readymade or may be prepared by mixing together 4 ounces of vaseline, ¼ ounce of carmine, and ½ ounce of aloes.

Cut the upper beaks of all chickens back to the flesh of the roof of the mouth. This is certain to be effective until the beaks grow out again.

Use deep litter and do not cover any more of the floor with wire mesh than is necessary to satisfy sanitation requirements.

Feed mash that provides proper amounts of protein for the age of the birds, and liberal amounts of ground oats or barley and wheat bran.

Attach one of the numerous types of antipick devices which are on the market to the beak or over the vent of each chicken in a laying flock. These are used extensively on poultry farms and are usually effective. Some types are adaptable to chickens from 3 to 4 months old.

EXTERNAL PARASITES

GENERAL CHARACTERISTICS OF MITES

Two distinct groups of mites attack poultry. One group is periodic, the parasite passing the greater portion of its life, not on the birds, but in cracks and crevices about the poultry houses from which it makes

nightly raids upon the roosting poultry to suck blood. The other group passes the entire life cycle on the birds, burrowing beneath the scales of the legs, into the skin at the bases of the feathers, into the shafts of the feathers themselves, or even penetrating the internal air sacs. These parasites may be distinguished from lice, by the facts that (1) an adult mite has four pairs of legs, a louse three pairs (a larval mite has three pairs of legs) and (2) the body of a mite is not divided into three distinct regions, that is, head, thorax, and abdomen.

THE COMMON POULTRY MITE

The common poultry mite, *Dermanyssus gallinae* (fig. 49, *A*), is a small mite. It is gray when unfed and reddish after having fed on blood, which it draws from the birds by means of long, piercing, styletlike mouth parts. When it is fully fed it is about the size of the head of a pin. This mite customarily hides away in cracks and crevices about the hen house during the day and migrates to the roosting birds at night to feed. It may, however, remain for considerable periods of time on very weak birds.

Symptoms and Diagnosis.—The common poultry mite is among the most injurious of all species attacking poultry. It causes extreme irritation to the birds, which in heavy infestations become droopy, weakened, progressively emaciated, and even die. The comb and wattles become pale as a consequence of the anemia caused by loss of blood. The irritation produced frequently causes laying hens to leave the nests. Egg production is greatly decreased and resistance to other diseases may be lowered. Infestations of this mite are especially serious in young chicks.

The presence of this mite may be detected by tiny, circular black and white dots ("pepper-and-salt marks") slightly smaller than flyspecks, caused by its excrement, on the roosts and along cracks, as well as by finding the mites themselves and their eggs in cracks and crevices, particularly those in the vicinity of the roosts.

Sources and Prevention of Infestation.—The common poultry mite is probably introduced into clean flocks most commonly in contaminated shipping coops. In some cases it may be brought in on introduced fowls, since young mites feeding for the first time may remain continuously on their hosts for as long as 3 days and 3 nights. In order to avoid the possibility of thus introducing an infestation, newly acquired birds should be quarantined in special coops for 3 days before being placed in the houses or with other chickens. By the end of this time the mites will have left the birds and be hiding in cracks in the coops. These coops should then be thoroughly disinfected with one of the recommended sprays or with

boiling water, or be destroyed by burning. Shipping coops from other poultry plants should not be left in or near clean houses, nor should secondhand equipment be introduced unless the proper precautions of disinfection are taken.

Infestations of this mite are sometimes acquired by the introduction of poultry into old houses. This is readily understandable when one recognizes that the mites may live for from 3 to 5 months without food.

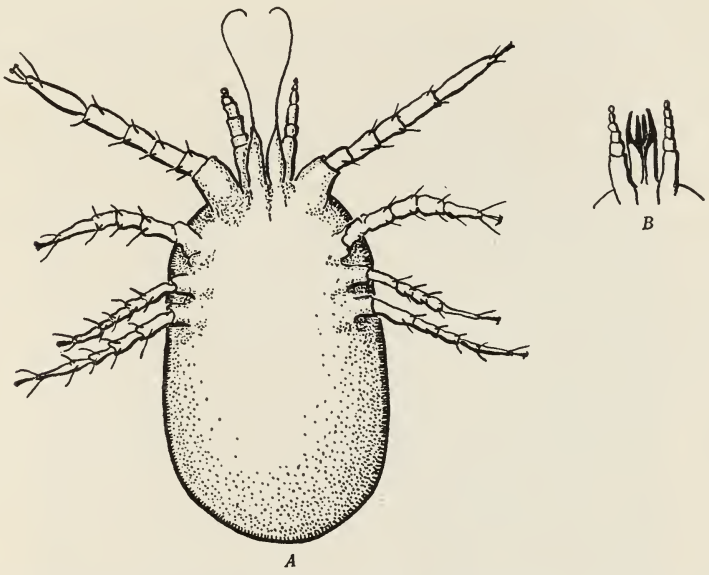

Fig. 49.—*A*, The common poultry mite, *Dermanyssus gallinae*; *B*, mouth parts of the tropical fowl mite, *Liponyssus bursa*. (From Ext. Cir. 8.)

Previously used vacant houses should be thoroughly cleaned, and sprayed with a recommended spray, before birds are introduced into them. All rubbish should be burned.

Wild birds, especially English sparrows, and rats and mice are acceptable substitute hosts and may introduce or maintain an infestation of the common poultry mite. Therefore, poultry houses and runs should be kept free of wild birds and rodents.

Control.—Cleanliness of the poultry houses and abundant sunlight are of paramount importance in the control of the common poultry mite. Since the mites are seldom found on the bodies of the birds during the day, except in the first feeding period or in dark nest boxes, control measures are directed most effectively against the hiding places such as cracks and crevices, beneath boards and boxes, and under droppings and other filth. As already indicated, a thorough cleanup of the premises

to which the birds have access, together with the elimination of every unnecessary article therein, such as boxes, coops, and boards, is the first step to be taken. Old nesting material should be burned, and, if the infestation is severe, roosts and nests should be dismantled and replaced if necessary by construction that will facilitate future cleanups. After the building has been cleaned, it should be sprayed, with special attention to the roosts and cracks and crevices of the floors and walls. In heavy infestations, the mites sometimes migrate to the outside of the house when the inside is sprayed. They should be looked for along the cracks on the outside and, if present there, a spraying of the outside is indicated.

The spray may be applied by bucket or knapsack spray pumps or by a power sprayer. A coarse spray is most effective and should be applied to each area from several different angles to insure penetration into all hiding places. There is considerable choice of sprays. The most satisfactory of these are anthracene oil, paraffin oil emulsion (p. 21), or crank-case oil to which from 10 to 15 per cent kerosene is added. The killing power of anthracene oil persists for several months, but sometimes it is advisable to make a second application, or more, if necessary, about a month later with a brush. Anthracene oil soaks into the wood rapidly without leaving a greasy residue to soil the feet, feathers, and eggs of the birds. The spraying of anthracene oil can be facilitated by adding to it about an equal part of kerosene. Crank-case oil or crude oil diluted with kerosene is efficient in killing the mites but does not possess the longevity of anthracene oil and leaves the roosts, floors, and walls in an oily condition which readily soils the birds and persons working in the houses. It should be remembered that the application of any highly inflammable substance to a wooden structure greatly increases the fire hazard and might present great difficulties in the collection of insurance in the event of a subsequent fire. Regardless of the spray employed, the birds should not be put back in until the houses have dried thoroughly.

Nicotine sulfate used on the roosts as described for louse control (p. 126) is effective in killing mites feeding on the birds and in protecting roosting birds. Used as a spray at the rate of 3 tablespoons per gallon of water, to which is added ½ teaspoon of baking soda, it gives very satisfactory results if applied carefully and in drenching quantities.

In those extreme cases where the control of the mite is impossible because of the character of the quarters, some relief may be afforded by painting the roosts thoroughly with anthracene oil or crude oil and wrapping the ends and other points of contact of the roosts with rags soaked in the same substance, to prevent the mites from gaining access to the fowls after they have roosted. To make this procedure effective, over-

crowding should be avoided and the back and the ends of the roosts should not be near enough to the wall to permit migration of the mites to the plumage of the birds. Obviously, rags soaked with an inflammable substance increase the fire hazard.

THE SCALY-LEG MITE

The scaly-leg mite, *Anemidocoptes mutans* (fig. 50), gets its name from its habit of burrowing beneath the scales of the shank of the leg,

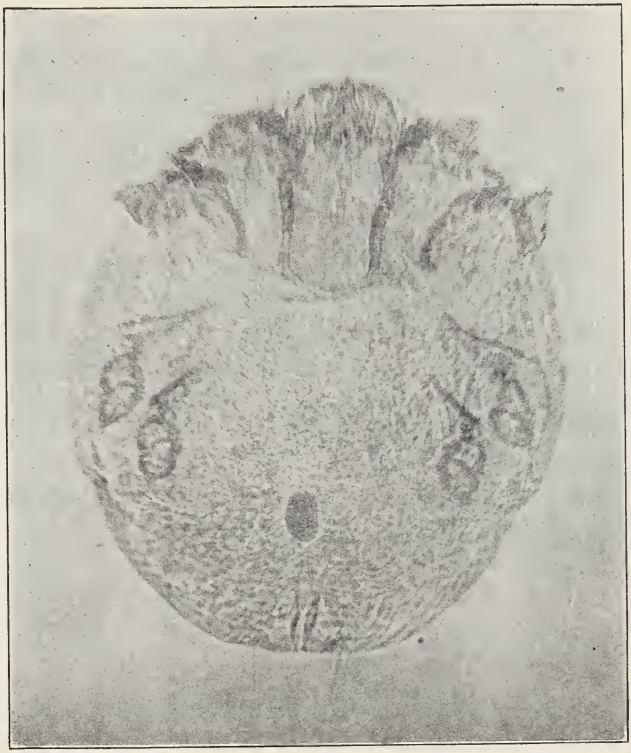

Fig. 50.—The scaly-leg mite, *Anemidocoptes mutans*, greatly enlarged. (From Ext. Cir. 8.)

where its presence and activities cause a lifting of the scales and a swollen condition of the shank. In extreme cases it produces such a pronounced distortion and deformity that the affected birds are unable to walk. Occasionally the comb and neck may be attacked. The mites usually begin their attack between the toes. The general physical condition of the birds is reduced in even moderate infestations. The scaly-leg mite is microscopic, but a diagnosis can usually be made from the symptoms. These symptoms are more commonly seen in the older birds.

Sources of Infestation.—Birds become infested with scaly-leg mites by the introduction of infested individuals into the flock or by being placed on infested premises.

Control.—There are several satisfactory treatments for scaly-leg. The legs may be dipped in crude petroleum or kerosene, preferably the former, or washed with soap and warm water to soften the thickened scales and crusts. Care should be taken to confine the oil to the affected parts of the leg. Then the scales are carefully removed by rubbing with the fingers. This must be done gently to avoid causing irritation and bleeding of the underlying skin. After the crusts are removed, the legs may be washed with some mild antiseptic and allowed to dry. After this the legs are smeared with an ointment containing 15 per cent sulfur or 2 per cent carbolic acid, or with a mixture of 1 ounce of Peruvian balsam and 3 ounces of ethyl (grain) alcohol. If carbolic acid ointment is used, it must be handled and stored carefully because of its highly poisonous nature.

A less thorough but also less laborious method of treatment is dipping the legs of the birds in a mixture of equal parts of crude oil and *raw* linseed oil, taking precaution to avoid wetting the legs above the scaly portion. If marked improvement is not noted, the treatment should be repeated in about 3 weeks; this is, however, seldom necessary.

Bathing the infested legs three times at 2- to 4-week intervals with a 0.5 per cent solution of sodium fluoride gives good results.

When the comb and neck are infested, they can be treated with the 15 per cent sulfur ointment previously mentioned.

Prevention.—In order to prevent spread of the infestation or reinfestation, the poultry house and particularly the perches should be sprayed under good pressure (150 to 300 pounds) with anthracene oil, crude petroleum, or paraffin oil emulsion (p. 21).

The legs of newly acquired birds should be observed carefully in order that infested birds may be segregated and treated before being placed with the clean flock.

THE DEPLUMING MITE

The depluming mite, *Cnemidocoptes laevis gallinae,* is a microscopic parasite which burrows into the skin at the bases of the feathers and produces an irritation which causes the birds to pluck out the feathers. This leaves nude scaly areas at the more severely infested places. The condition is known as depluming mange, or scabies. The stumps of feathers examined soon after the quill is broken will be observed to be surrounded by scales or crusts, and adjoining feathers will be found to be similarly affected. In this way depluming mange can be differentiated from the feather-pulling vice discussed on page 112.

Depluming mange usually begins in the spring and spreads rapidly through the flock as the weather becomes warmer. The loss of feathers ordinarily begins on the rump near the insertion of the tail feathers and spreads to the back, neck, head, breast, and thighs; only rarely are the large tail and wing feathers lost. Egg production and weight gains may be decreased. Male birds seem to be more susceptible than females.

Sources of Infestation.—Depluming-mite infestations are introduced into a flock with the acquisition of newly infested birds or by placing birds in houses or runs previously infested and not thoroughly cleaned before the new birds are introduced. Spread is favored by any circumstances, such as copulation, which brings about bodily contact.

Control.—Depluming-mite infestations may be satisfactorily treated by thoroughly soaking the birds in a dip composed of 10 pounds of finely ground (350- to 500-mesh) wettable sulfur in 100 gallons of water on the morning of a warm day when there is little wind. If the birds are infested with lice, these parasites may be controlled at the same time by the addition of ¾ ounce of commercial sodium fluoride to each gallon of dip. When only a few birds are involved, the affected parts may be treated with an ointment made by thoroughly mixing 1 part of finely ground sulfur with 4 parts of lard, vaseline, or lanolin.

Prevention.—Care should be exercised not to introduce infested birds into a clean flock. Spread of the infestation can be prevented by treating the birds as described above and at the same time cleaning the poultry houses and spraying them as described in the discussion on the prevention of scaly-leg. Infested cocks should not be used in breeding flocks until they have been effectively treated because these birds may transmit the infestation during treading.

THE TROPICAL FOWL MITE AND THE FEATHER MITE, OR NORTHERN FOWL MITE

The tropical fowl mite, *Liponyssus bursa* (fig. 49, *B,* p. 115), and the feather mite, or northern fowl mite, *Liponyssus sylviarum,* are very closely related species which can be distinguished from one another only by microscopic examination. They stay on the hosts or in the nest and breed in both places, a habit which aids in differentiating them from the common poultry mite (p. 114), which customarily attacks the birds only for short periods in order to feed and spends the rest of the time in cracks about the roost, where it breeds. The entire life cycle of the tropical and the northern fowl mite from egg to adult requires only 8 to 12 days.

Symptoms and Diagnosis.—Since these mites hatch from eggs on feathers attached to the birds or in the nest, and do not have to seek a

host and since they grow to adults so fast, their multiplication is exceedingly rapid, and infestations with them may quickly become very severe.

The mites are most numerous about the vent and tend to accumulate on a few rather than many feathers, although in heavy infestations they may be generally distributed over the body. These parasites give the feathers a dirty appearance, and the skin in invaded areas becomes scabby due to the irritation produced while they are sucking blood. An untreated heavy infestation may result in death.

Sources of Infestation.—Infestations with these two species of mites occur as the result of the introduction of infested birds into the flock, placing birds in buildings in which an infestation already exists, or by close contact with English sparrows or their nests.

Control.—The nicotine sulfate treatment described for the control of lice (p. 126) will control these mites satisfactorily but should be applied three times at intervals of 3 days instead of twice at intervals of 10 days as recommended for lice. If necessary such a treatment should be repeated still again after an interval of 2 weeks. A very satisfactory treatment is to dip thoroughly each well-feathered bird in a solution made up of 1 gallon of water and 2 ounces of finely ground wettable sulfur. The dipping should be done in the morning of a warm sunny day when little wind is blowing or in a heated building, and the dip should be lukewarm to reduce shock. The head should be submerged for an instant. In cold weather fair results may be obtained by thoroughly dusting the adult birds with wettable sulfur or pyrethrum. It does not seem advisable to dust chicks with sulfur but pyrethrum may be used on them.

At the same time the birds are treated, regardless of the chemical being used or its method of application, the building should be thoroughly cleaned, all nesting material and litter being removed and burned, and the building sprayed completely with anthracene oil, paraffin-oil emulsion (p. 21), or crude petroleum. The area over which the litter is transported for burning and around which it is burned and the implements and vehicles used in transporting the litter should also be sprayed.

Since English sparrows harbor these parasites, their nests located in and around poultry houses and runs should be destroyed by burning and the area immediately around them should be sprayed.

Prevention.—Care should be taken not to introduce infested birds into the flock and constant vigilance should be exercised to prevent English sparrows from nesting in or adjacent to poultry houses. Clean birds should not be placed in buildings until the buildings are known to be free of these parasites. This may be done by placing a few birds in such buildings and carefully examining them for mites a few hours later or by making a minute examination of cracks and crevices.

THE AIR-SAC MITE

The air-sac mite, *Cytodites nudus,* is a small oval mite which lives in colonies in the respiratory organs and particularly in the air sacs, where they can be seen as very minute yellow spots. They are able to penetrate not only into the tracheae and bronchi, but also into all parts of the lungs, the air sacs, and even into the air-containing cavities of the bones. Clinical symptoms have rarely been observed even when the parasites were very abundant. Some authors, however, have attributed clinical symptoms similar to tuberculosis or "going light" to heavy infestations of these mites, and at least one case of obstruction of the bronchi with consequent asphyxiation has been observed. The life cycle of this species is unknown.

A definite diagnosis of air-sac-mite infestation can be made only at autopsy.

No treatment is known for this infestation.

THE SUBCUTANEOUS MITE OF FOWL

The subcutaneous mite of fowl, *Laminosioptes cysticola,* has a long, slender body and bears a line of division between its fused head and thorax and its abdomen. This mite invades subcutaneous tissue, particularly in those regions of the body where the skin is loose, such as the neck, the breast, the flank, around the vent, and the thighs. There it causes small, flat, oval nodules, which resemble bits of fat. When the mites die, they become surrounded with caseous or calcified material. These mites apparently do no harm to the host but may reduce the marketable value of birds intended for human consumption. The life cycle of the mite and its treatment and prevention are unknown.

THE QUILL MITE

The quill mite, *Syringophilus bipectinatus,* has been taken from poultry in California. This mite feeds on the quills, apparently chiefly or only on those of feathers about to be dropped, and is generally believed to be of no economic importance. In some instances, however, a peculiar molt, involving over half the body, is produced. The mites are found in a yellowish-gray or brownish powder in the quills but usually can be detected only under a microscope.

DIFFERENTIATING TICKS FROM MITES

Ticks are closely related to mites, but they are always larger, easily visible to the naked eye, and have a thick leathery cuticula, or skin. They may be distinguished from mites also by the possession of a pair of

breathing-pore plates situated on the side above, and usually behind, the fourth pair of legs. Like mites, ticks have three pairs of legs in the larval stage and four pairs in the adult.

THE FOWL TICK, OR BLUE BUG

The fowl tick, or blue bug, *Argas persicus* (fig. 51), is the only tick attacking fowls that is of economic importance. The adult tick is flat, egg-shaped in outline, dark brown in color, from ¼ to 7/16 inch in length, and about half as wide at its widest part. The small, spherical, brown

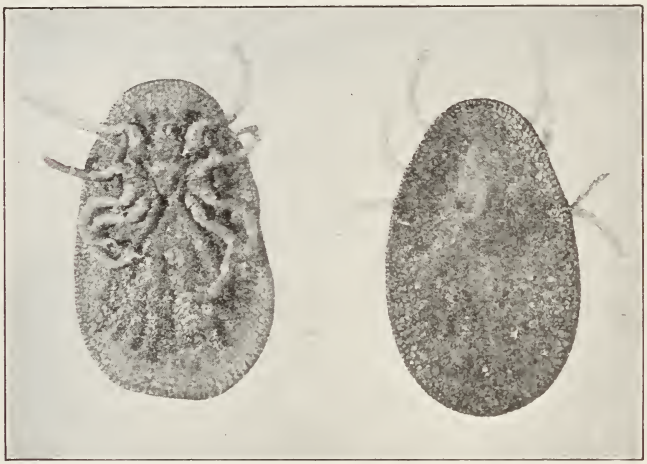

Fig. 51.—Ventral and dorsal views of the fowl tick, or blue bug, *Argas persicus*, enlarged. (From Ext. Cir. 8.)

eggs are laid in batches of from 20 to 100, in cracks and crevices of the poultry house and under the bark of trees. A total of from 500 to 900 eggs are laid by each female. These eggs hatch in 3 weeks or more, and the emerging larvae migrate, usually at night, to the birds and attach themselves on those parts of the body where the feathers are not very dense, especially under the wings, and gorge themselves with blood. Complete engorgement requires about 5 days, rarely as long as 10 days. After this the larvae drop off their hosts and hide away to transform to the nymphal stage, which occurs after about 7 days. There are two nymphal stages, each of which lasts about 2 weeks, in the life cycle of this tick. It feeds but once for about 2 hours during each of these stages.

The nymphs, like the adults, attack their hosts only at night and hide away in sheltered spots during the day. The adults feed approximately once a month for a period of about 2 hours, and the females deposit a batch of eggs after each meal.

This tick is able to live for long periods without food. Adults and nymphs are known to have survived nearly two and a half years of starvation, and it is highly probable that in vacant infested poultry houses some of them may successfully withstand fasting considerably longer. The larvae, however, are not so resistant to the effects of starvation, usually dying in about 3 months if they do not find a host. This ability to exist for considerable periods of time without access to food makes control of the parasite by starvation, through excluding all birds from the houses, a very difficult matter, and explains the infestation of clean birds which have been introduced in houses that have been vacant for a year or more.

The fowl tick usually appears to be more abundant in the spring months. This impression is created because the various stages of development are greatly retarded during the winter months. In the spring the ticks resume the normal developmental rate, which continues during the hot, dry summer months.

Symptoms and Diagnosis.—Birds attacked by considerable numbers of the fowl tick show a weakness of the limbs, extreme emaciation, and paleness about the head. Egg production is greatly reduced or may entirely cease. The growth of young birds is stunted. Sitting hens are very seriously annoyed by the attacks of this parasite. In heavy infestations, chickens lose vitality and may die from anemia, which is possibly supplemented by a poisonous secretion from the ticks. Young chicks are easily killed by the effects of the ticks.

The fowl tick transmits fowl spirochaetosis, a fatal disease caused by *Treponema gallinarum,* and poultry piroplasmosis, caused by *Aegyptianella pullorum,* in those parts of the world where these diseases occur. These diseases have not been reported from California.

Sources of Infestation.—Fowl-tick infestations are acquired by placing birds in infested buildings, by introducing infested birds into the flock, and, less frequently, by contact with domestic or wild ducks, geese, and turkeys.

Control.—Since the larval ticks remain on their hosts for a considerable time, sometimes as long as 10 days, it is highly advisable to treat the birds as well as the buildings and runs. When flocks are small enough, the birds should be placed in wooden crates, the cracks of which afford ample hiding places for the ticks after they drop off the hosts, and kept there for 10 days. In the meanwhile the buildings and runs should be thoroughly cleaned of all rubbish and loose boards and the loose bark of immediately adjacent trees removed. All of this debris is burned. If the buildings are old and worthless, they are best burned. Buildings are thoroughly sprayed with paraffin-oil emulsion (p. 21) or anthracene oil

at a good pressure. The trunks and lower limbs of nearby trees should also be sprayed. It is well, when the buildings have dried after spraying, to eliminate hiding places as far as possible by closing all cracks with hot tar. If the cleaning and spraying is not thoroughly done, the ticks may not be exterminated, and then it is necessary to clean and spray the building a second time after an interval of 10 days.

After the birds have been kept in crates for 10 days, they are removed and returned to the clean buildings, and the crates are burned or disinfected with the spray used on the buildings or with scalding water.

If only a few fowls are infested with larval ticks, they may be dipped in a 2 per cent solution of creolin; the same procedure is followed as for tropical fowl mite (p. 120). This practice is also advisable in birds showing signs of weakness resulting from an existing infestation of larval ticks.

Prevention.—Birds should not be placed in buildings previously occupied by poultry, even though they may have been vacant for as long as two and a half years or even longer, unless they are definitely known to be free of ticks. This can be determined by placing a few white birds in the buildings and examining them for ticks during the night. New birds should not be introduced into the flock before they have been very thoroughly inspected for tick infestation or been quarantined in suitable crates. If reinfestation is probable and no other prevention is feasible, a considerable degree of protection may be afforded the birds by suspending the perches from the roof by wires which pass through small cups of oil and arranging the roosts so that they have no contact with the walls. This prevents the ticks from crawling onto the perches.

LICE

Lice can be distinguished from all other parasites on the body of poultry by the facts that they have three pairs of legs in all stages and their bodies are divided into three sections, the head, thorax, the abdomen, and flattened dorsoventrally, that is, as if a flattening force had been applied on their backs. They seldom are over $\frac{3}{16}$ inch in length and are of a yellow or grayish color, sometimes ornamented with dark stripes (fig. 52). They are never uniformly dark brown or red; parasites of these colors may be fleas, mites, or ticks.

The entire life of lice, including the egg stage, is spent on the body of the birds. It is only by accident that the parasites leave their hosts except to migrate to other hosts of the same species. The eggs require about a week to hatch, after which maturity is reached in about 2 weeks.

It is not unusual to find at least six of the common species of lice on a single bird.

The shaft louse, *Menopon gallinae,* and the body louse, *Eomenacanthus stramineum,* are perhaps the commonest poultry lice. They may be separated from all other chicken lice by the fact that their antennae are buried in depressions and are not outstanding from the head. They are both pale yellow in color, but the former has one row of bristles on the back of each abdominal segment while the latter has two rows.

The wing louse, *Lipeurus caponis,* and the head louse, *L. heterographus,* have antennae that stand out, and the combined second and third leg-bearing segments called the "pterothorax," from which the last two pairs of legs originate, is square-sided. In the wing louse the

Fig. 52.—Three common species of poultry lice. (From Ext. Cir. 8.)

forehead, the area just in front of the antennae, is nearly straight-sided, while in the head louse the sides of the forehead converge rapidly. The posterior abdominal segments of the females vary in the amount of heavily pigmented margins.

The fluff louse, the smallest of the common poultry lice ($\frac{1}{20}$ inch) is known as *Goniocotes hologaster*. The thorax is roughly triangular in appearance. The antennae are outstanding and slender.

Symptoms and Diagnosis.—A few lice may cause no detectable damage and produce no symptoms. Under suitable conditions, however, they may multiply with amazing rapidity and therefore even a few lice are potentially of considerable importance. They irritate the birds to such an extent that they become very restless and neither feed nor sleep well. Heavily infested hosts are in poor condition and show damaged plumage. Lousiness results, in severe infestations, in marked decrease or cessation of egg production, loss of weight or failure to gain properly, possible loss of resistance to other diseases, and, with young chicks, even in death. The head louse and the body louse ordinarily exert the most serious influence upon the host of any of the species of lice. Diagnoses

are very easily made by carefully inspecting the birds and detecting the lice on the feathers or body.

Source of Infestation.—Louse infestations are acquired from contact with infested birds. The parasites crawl from infested hosts to noninfested ones when they come in close contact with one another. Lice cannot live long off a host.

Control.—A highly effective and easily applied method of treating chickens for all species of lice except the head louse consists of applying Black Leaf 40 to the roosts in a thin layer (about ½ pound per 100 feet of roosts) just before roosting time. The back and ends of the house should be closed but the front should be partially open to avoid the possibility of injury to the birds from the fumes. The flock should be inspected during the first 2 evenings after treatment, and all birds placed on the roost that are found roosting elsewhere. The warmth of the birds' bodies causes the chemical to evaporate, permeate the feathers, and kill the lice by fumigation. Perfect control from a single treatment has been obtained in many instances even when the temperature during the night has approached freezing. Failures that have occurred are probably attributable to windy or drafty locations of the roosts or an unfavorable atmospheric condition of an, as yet, unknown character. Since unharmed louse eggs have been found on treated birds, a repetition of the treatment in 10 days is advisable. In lieu of this, the birds should be closely observed for at least 2 weeks and treated again if any young lice appear. Tests with a solution of pure nicotine sulfate conforming to Black Leaf 40 in hydrogen-ion concentration and content of nicotine sulfate have invariably failed to destroy the lice. This would indicate that the effectiveness of Black Leaf 40 is not due solely to the nicotine sulfate it contains. Under no circumstances should the treatment be applied when roosts are freshly whitewashed nor should a preparation of pure nicotine (nicotine alkaloid) be substituted.

Sodium fluoride (commercial grade) is a highly effective chemical for the individual treatment of chickens for lice. It is a stomach poison for lice and remains on the bodies of the birds long enough to kill the young lice that hatch from eggs which were on the feathers when the birds were treated. There are three methods of application, the pinch method, dusting, and dipping. The pinch method consists in rubbing pinches of sodium fluoride on the skin, not merely on the feathers, as follows: one on the head, one on the neck, two on the back, one on the breast, one below the vent, one on the tail, one on each thigh, and one on the underside of each wing. This is the best method to use for head lice on old and weak or young birds. The chemical should be applied cautiously and in very small pinches to young birds.

In the dusting method, the sodium fluoride is diluted with 3 parts of flour or talc. Then the bird is inverted and the dust is applied liberally with a shaker while thoroughly ruffling the feathers.

Dipping is the most economical and rapid method of applying sodium fluoride and is most suitable for large flocks. The dip does not irritate the respiratory tract of the operator as does the powder in the pinch or dusting method. To make the dip, 1 ounce of commercial sodium fluoride is dissolved in 1 gallon of warm water. Dipping should be done only on the morning of a warm day. The bird should be inserted into the dip feet first, the feathers should be ruffled to permit the dip to penetrate to the skin, and the head should be ducked once or twice before taking the bird out.

One pound of sodium fluoride will treat 100 birds by the pinch method and 200 by dipping.

Sodium fluosilicate, finely ground sulfur, or a dusting powder made of 3 parts benzine and 1 part carbolic acid, to which is added sufficient plaster of paris to absorb the liquid, applied in the same way as described for sodium fluoride dusting, gives good results. It must be remembered, however, that carbolic acid is very poisonous.

Mercurial, or blue, ointment, if rubbed on the skin just below the vent, a quantity about the size of a pea being used, will kill body lice going to the vent for moisture. Mercurial ointment, however, is very poisonous and consequently must be handled with care.

Head lice on young chicks can be effectively controlled by the application of cottonseed or any other bland oil or soft fat to the infested region.

Prevention.—Louse infestations may be prevented by carefully inspecting all new birds before they are introduced into the flock and quarantining and treating if necessary, and by otherwise avoiding contact with infested birds. Excessive infestation may be prevented to some extent by supplying the birds with ample dusting boxes filled with fine road dust to which sodium fluosilicate has been added at the rate of 1 part of the chemical to 3 parts of dust. This latter procedure should be regarded merely as an adjunct to the more effective practices described above and should not be relied upon too heavily. Spraying the house and roosts has no effect on louse infestations. Brooding hens, unless definitely known to be free of lice, should be treated before being placed with newly hatched chicks.

GENERAL CHARACTERISTICS OF FLEAS

Fleas may be distinguished by their dark brown color, the presence of three pairs of long legs adapted for jumping, the absence of wings, and the laterally flattened body. Three species of fleas are known to attack

poultry; but one of these, the European hen flea, *Ceratophyllus gallinae,* has not been reported from western North America and hence will not be discussed in this bulletin.

THE STICKTIGHT FLEA

Although the sticktight flea, *Echidnophaga gallinacea,* is parasitic on dogs, cats, rats, and other wild mammals, as well as several species of wild birds, it habitually attacks poultry and finds this host admirably suited for its purposes. The sticktight flea, unlike most other species of fleas, remains tightly attached in clusters to the host throughout adult life. The eggs laid by the attached females ordinarily fall to the ground and hatch. In rare instances the eggs are retained in the burrows or ulcers caused by the imbedded females. In such cases the larvae upon hatching usually fall to the ground, but they may remain in the tissues, where they produce a condition resembling the invasion of tissues by blowfly maggots. These larvae are tiny, white, caterpillarlike organisms with chewing mouth parts. They subsist on blood which is constantly being evacuated by the adult fleas and falls with the eggs to the ground. When the larvae become full grown, they spin a whitish cocoon, within which they change from the larval stage to that of an adult, after which they attach themselves to the skin of their host and suck blood.

Symptoms and Diagnosis.—On chickens the sticktight flea is confined to the face, ear lobes, wattles, and comb and is most frequently attached about the eyelids and comb. The constant irritation, particularly in spots where the fleas are closely aggregated and present in large numbers, together with the slight burrowing activity of the insects, causes the formation of ulcers so extensive at times that blindness and subsequent death are produced. Young fowls when heavily infested often die quickly. Even mild infestations reduce egg production and retard growth. Diagnosis is made by finding adult fleas on the hosts or larvae in the litter.

Sources of Infestation.—Sticktight-flea infestations are acquired by contact, direct or indirect, with infested poultry, wild birds such as quail, Brewer's blackbirds, and English sparrows, rats, mice, dogs, and cats, and by the introduction of chickens into infested buildings. Under dry and cool conditions, these fleas may remain alive for some months in vacant poultry buildings and runs and may be maintained in considerable numbers in such places by hosts other than poultry.

Control.—The first step in the control of this parasite is thorough cleaning of the infested buildings and pens and the burning of all rubbish collected. This is followed by a thorough spraying with paraffin-oil emulsion (p. 21), crude oil, kerosene, kerosene emulsion (p. 21), or a reliable proprietary pyrethrum spray fortified with lauryl thiocyanate.

These fleas cannot thrive in damp places and if the yards are sprinkled two or three times a week, for a week or two, breeding is greatly reduced. One of the most efficient methods of preventing breeding is to scatter salt freely about the yards and then wet the soil thoroughly. Fowls must not be permitted to eat the salt because it is poisonous to them.

The sticktight flea can be killed on the birds by the application of 2 per cent carbolic ointment, mercuric ointment, sulfur mixed with 5 parts of a bland oil, an ointment made up of 1 part kerosene and 2 parts lard, or tincture of iodine. Great care must be exercised, however, not to get any of the ointments in the birds' eyes, for they may produce blindness. The application of these ointments must be confined to the seats of infestation.

All manure and other scrapings from the poultry houses and yards that are to be used for fertilizing purposes should be turned under. The mere storing of the manure outside of the house in piles or spreading it on fields will not prevent the development of the flea larvae to the adult stage, although it will prevent them from gaining direct access to confined fowls; the cats, dogs, and men of the neighborhood will be the alternative recipients of their attention unless the manure is carefully turned under by plowing or spading.

Prevention.—The exclusion of wild birds, dogs, and cats from poultry houses and runs and rigid rat and mouse control are very essential in the prevention of sticktight-flea infestations. New birds should not be introduced into the flock until it has been definitely ascertained that they are free of this parasite and care should be taken to determine that buildings into which poultry are to be placed are free of fleas.

THE WESTERN HEN FLEA

The western hen flea, *Ceratophyllus niger,* may at times be a serious pest of poultry. This flea is considerably larger than the sticktight flea and attaches itself to its host only for short periods of time to suck blood, usually leaving the host's body between meals and spending its time in the litter of the nests, although it may occasionally remain unattached on the host. It is a voracious feeder and attacks men more readily than does the sticktight flea. The habits are otherwise like those of the sticktight flea.

Symptoms and Diagnosis.—Birds infested with the western hen flea show a decrease in egg production. In heavily infested flocks, laying may cease entirely and the birds become emaciated. In especially severe cases, death may result. Young birds appear to be more severely affected than older ones.

A diagnosis is most readily made by finding the fleas in the litter.

Control.—The control methods are the same as those described for sticktight-flea infestations with the exception that ointments are never applied since the parasites are not permanently attached and do not appear in clusters on the host.

Prevention.—The preventive practices are the same as those described for the sticktight flea.

THE COMMON HOUSE FLY

The common house fly, *Musca domestica,* lays its eggs preferably on freshly deposited manure, but it will also utilize for this purpose decomposing vegetable and animal matter or even moist soil saturated with nitrogenous material. When garbage is fed, unless extra precautions are taken, particularly heavy fly breeding will occur because of the attractiveness of this material to flies. These eggs hatch in about 2 days into tiny, glistening white, footless maggots that grow rapidly for approximately a week until about ½ inch in length, when they migrate from the moister part of the medium in which they are developing to a drier portion or to surrounding soil; here they transform into brown, barrel-shaped pupae, from which they emerge as full-grown flies in about 4 days.

House-fly control around poultry plants is important not only because of the annoyance these insects cause man and of the possibility of carrying filth and disease-producing organisms to his fowls but also because they serve as intermediate hosts for some of the poultry tapeworms.

Control.—Removal of droppings and moist litter from the poultry houses and yards at intervals of less than 7 days is essential in controlling house flies on poultry ranches.

Where poultry manure has a definite cash value, the droppings (scrapings from the droppings board) and the sweepings (material from floors and yards) are segregated in fly-tight bins and collected at frequent intervals.

Many producers accomplish the same results during the dry season by spreading the manure daily on plots to be fertilized, in a thin layer that will dry out rapidly. The fly larvae will be destroyed by the drying, but the manure should be plowed under about once a week where flea infestations exist, to avoid the emergence of these latter insects.

The maggot trap is another effective method of fly control where poultry manure is concerned. This consists of a concrete pan about 9 feet long, 12 feet wide, 6 inches deep, and with walls 4 inches thick. This is kept filled with water to which a film of oil is added. A table with legs 1½ feet high and slightly smaller in area than that of the pan is placed in the pan. The top of the table is made of crossed inch laths spaced

their own width apart, leaving a latticed appearance, and covered with straw to prevent the manure from falling through. The manure is then placed on this table or rack and moistened enough to make it thoroughly wet but not dripping. Each addition of excreta is treated in the same way, the whole deposit kept wet at all times. The fly larvae flourish under this treatment, but when ready to pupate their search for a dry place ends invariably in a fall to the oil-filmed water in the pan below. If the manure is collected regularly and placed on the maggot trap and if the mass of manure is kept wet, this method will return 100 per cent efficiency.

Dead fowls should be disposed of promptly through the garbage-collection system in cities and towns or by incineration in rural areas. If it is necessary to bury dead birds, or other dead animals, a hole at least 1½ feet deep should be prepared, the body covered with a crude oil, and the ground well tamped.

Adequate, flyproof storage receptacles for kitchen refuse are essential in the prevention of fly breeding. The addition of a small quantity of turpentine to vessels containing garbage acts as a repellent and is harmless. Where there is no effective garbage-collection service, the garbage should not be permitted to accumulate beyond reasonable limits.

Septic tanks should be kept in a condition of good repair and kept tightly sealed in order that flies may not have access to their contents and breed in them.

Particular care should be taken to the feeding of wet mash and liquid milk in order to prevent their accumulating and becoming a source of fly breeding.

Creosote oil sprayed on fences, walls, and floors will repel adult flies, but the penetrating odor is disagreeable, although it is less messy than crude oil or fuel oil and does not present the fire hazard of the latter materials.

If the manure cannot be disposed of promptly and adequately, poisons may be applied to kill the fly larvae, but most of these materials interfere to a greater or lesser degree with the fertilizing value.

Powdered hellebore (powdered roots of *Veratrum album* or *V. viride*) is employed in the chemical treatment of manure to prevent fly breeding. One-half pound of powdered hellebore is stirred in 10 gallons of water and allowed to stand for 24 hours before using. This solution is spread over the manure at the rate of 10 gallons to each 10 cubic feet of refuse material. The hellebore solution is somewhat poisonous and livestock should be prevented from drinking it. Poultry, however, are not poisoned by pecking at hellebore-treated material; but still it is not desirable, for other and obvious reasons, for them to have free access to manure piles.

No injury to plants appears to result from fertilization with hellebore-treated manure.

Powdered borax, which has the advantages of being inexpensive, comparatively nonpoisonous, and noninflammable, is effective in preventing fly breeding in manure piles. Three-quarters of a pound of powdered borax is dissolved in 10 gallons of water and sprinkled over the manure at the same rate as given above for the powdered hellebore treatment. It is inadvisable, however, to utilize borax-treated manure as fertilizer repeatedly on soils because this may create too high a boron content.

Creosote oil may be sprayed on each daily addition to the manure pile at the rate of 1 gallon to the ton of excreta if it is not to be used as fertilizer.

Electric fly traps have come into widespread use and are probably more efficient than any other device for catching flies, but it should be recognized that even though large numbers of adult flies may be captured, this is only a palliative measure, and that no real relief from these pests can be obtained unless the control measures are directed against the breeding places. Only electric traps approved by the Underwriter's Laboratory should be employed, in order to avoid accidental burns or electrocutions and fires.

Poisoned fly baits are sometimes used, but these, like the electric traps, are only palliative since they do not control the breeding places. Perhaps the most commonly used of these is formalin, which is poisonous to higher animals and man as well as to flies. A pint of a 1½ to 2 per cent solution, is added to 1 pint of diluted canned milk, 1 pound of sugar, and 3 gallons of water. A small piece of bread or sponge is placed in a shallow dish and saturated with the formalinized bait. The bait must be replenished regularly and frequently.

Another effective poison bait, which is used in the same way as the formalin solution, is composed of 3 tablespoons of sodium salicylate dissolved in 1 pint of water.

WOUND-INVADING MAGGOTS

A number of species of flies, the most important of which are the blowflies (*Lucilia* species), the wool maggot (*Phormia regina*), the primary screwworm (*Cochliomyia americana*), and the secondary screwworm (*C. macellaria*), produce larvae which invade wounds, both large and small, and thereby cause serious damage. The blowflies and the wool maggots will deposit their eggs on or adjacent to either fresh or old wounds. The primary screwworm has a very strong preference for fresh wounds. The secondary screwworm prefers older wounds in which some necrosis has occurred.

Symptoms and Diagnosis.—The larvae hatching from the eggs deposited on or adjacent to wounds burrow into and feed upon the tissues of the host. They very quickly create extensive damage and frequently produce death unless effective treatment is promptly initiated. Maggots hatching in feces which have collected on the feathers around the vent may invade the cloaca, penetrate into the abdominal cavity, and cause a fatal peritonitis. Diagnosis is made by the examination of wounds, which when infested typically have a bloody exudate, and detection of the maggots, which may be so deeply imbedded in the tissues that only the flat posterior tip of their bodies are visible.

Treatment.—Maggot-infested wounds should be cleaned by gentle swabbing with absorbent cotton and then irrigated with commercial benzol or a mixture of 15 parts chloroform and 85 parts bland oil (olive oil, cottonseed oil, or liquid petroleum). When benzol is used, a small amount is sprayed on the wound to stop the flow of blood and serum; then more benzol is added and the wound is plugged with cotton. The plug is kept in place for at least 3 minutes to allow the fumes to penetrate and kill the maggots. The chloroform-oil solution will ordinarily cause most of the maggots to leave the recesses of the wound before they die; the benzol will do this to a lesser degree. As many dead larvae remaining in the wound should be removed with blunt-pointed forceps as is possible without causing bleeding. The wound is then covered with diphenylamine.

Smear no. 62, which contains 35 per cent benzol, 35 per cent diphenylamine, 10 per cent turkey-red oil, and 20 per cent lampblack, will kill older maggots in the wound as well as prevent younger ones from developing; but when this material is employed the dead maggots are left in the wound, unless they migrate to the surface before dying.

Cloacal infestations are best treated by irrigation with small quantities of carbon tetrachloride.

Prevention.—In order to prevent maggot invasion, the flock should be watched for birds with wounds or collections of feces on the feathers beneath the vent. Injured birds should be kept out of the flock until the wounds are healed. Manure should be removed from the feathers around the vent and the birds in which such contamination occurs should be kept out of the flock until it is discovered that no more fecal material will collect.

Noninfested wounds should be coated with Smear no. 62 or covered with diphenylamine, which must be ground sufficiently fine so that at least 50 per cent will pass through a screen having 40 meshes to the inch. Diphenylamine has a tendency to cake, especially when the containers are exposed for a time to high temperatures. When this occurs, regrind-

ing to its original fineness is necessary before the material can be used. Storage in a cool place in tight metal or wooden containers will aid in preventing excessive caking of the chemical.

Sharp objects, such as splintered ends of boards or ends of wire in houses, on which chickens can be readily injured should be avoided. Care should be taken not to frighten the chickens by sudden entrance, especially of strangers, into houses or when catching them, for this may cause them to fly against side walls, wire-mesh fronts, or pieces of equipment.

Wound-invading flies should be controlled by the disposal of dead birds. All dead bodies, whether or not they are infested with maggots, must be promptly disposed of by burning or thoroughly poisoning with arsenic and burying at a depth of at least 2½ feet.

THE COMMON BEDBUG AND THE MEXICAN CHICKEN BUG

The true bugs which attack poultry may be distinguished from other insects by the flattened body, elongate oval shape, yellowish-brown to dark-brown color, long 4-jointed antennae, and vestigial wings. The compound eyes protrude conspicuously from the sides of the head and the first thoracic segment is large and deeply notched anteriorly where the head is inserted. The abdomen has eight visible segments, and the entire body is covered with bristles and hairs. On the ventral side of the thorax are stink glands, which impart a characteristic odor to these insects.

In California only two species of this group, the common bedbug (*Cimex lectularius*) and the Mexican chicken bug (*Haematosiphon inodorus*), are known to attack poultry. These bugs hide away in cracks and crevices near the sleeping places of their hosts and usually suck blood only at night. They will, however, bite setting hens in the daytime. They can successfully withstand starvation for at least several months. Under favorable conditions they are able to multiply very rapidly.

Symptoms and Diagnosis.—These insects at times cause severe irritation and anemia in poultry. Their presence may be detected by the careful examination of cracks and crevices, particularly those near the roosts and nesting boxes.

Sources of Infestation.—The bugs gain access to poultry houses from adjacent infested human habitations and from visiting wild birds.

Control.—Poultry houses should be thoroughly cleaned and all rubbish removed and burned. The cracks and crevices should be thoroughly sprayed with paraffin-oil emulsion (p. 21), or a good commercial pyrethrum spray fortified with lauryl thiocyanate. Also, it is well to close all such hiding places by painting them with hot tar.

Prevention.—All buildings should be determined to be free of these parasites before birds are placed in them. Second-hand equipment should be carefully inspected, and treated if necessary, before being placed in clean houses. Infested human habitations on poultry farms should be ridded of bedbugs.

INTERNAL PARASITES

FLUKES

Flukes are unsegmented worms which are flattened and leaflike in most cases. They generally possess one or two cup-shaped suckers, which are organs of attachment; when two suckers are present, one is anterior and the other is ventral or posterior. All of the species known to normally parasitize poultry are hermaphroditic. Their eggs, which are eliminated from the body of the fowl with the feces, hatch in the presence of moisture. The larval forms that emerge seek an intermediate host, an appropriate species of snail, in which they grow and then divide into a varying number of daughter larvae. These latter forms migrate from the body of the snail and, in the case of the poultry flukes here considered, penetrate the bodies of second intermediate hosts, such as tadpoles and dragonfly nymphs, where they await until these hosts are eaten by the birds.

Echinoparyphium recurvatum, the only fluke that has been reported from poultry in California, is a small leechlike intestinal fluke, ¼ inch long, which attaches itself to the lining of the small intestine. It is characterized by a collar of heavy spines about the sucker at the anterior end of the body. The eggs of this parasite escape from the body of the host in the feces and hatch, when they come in contact with water, into swimming embryos which migrate through the water to appropriate snails, in whose bodies a multiplication occurs. The still immature, but more advanced, forms escape from the intermediate host and migrate to tadpoles, in the kidneys of which they encyst. Fowls acquire the flukes, still in an immature stage, by eating infected tadpoles.

Symptoms and Diagnosis.—Fowls infected with *Echinoparyphium recurvatum* become emaciated, anemic, and sometimes show weakness of the legs. Post-mortem examinations reveal a marked inflammation and swelling of the intestinal lining. In heavy infections there is frequently a fatal termination. Diagnosis is made by finding the worms in the intestine at autopsy or by detecting the presence of the eggs in the droppings by microscopic examination.

Treatment.—Carbon tetrachloride or tetrachlorethylene is administered in doses of from 1 to 2 cc, according to the weight of the bird. The drug is best administered with about 3 cc of paraffin oil by means of a syringe and a piece of narrow, flexible rubber tubing about 10 cm

long which is gently pushed down the esophagus into the crop. It may also be given in gelatin capsules, but great care must be taken that these capsules are not broken in the mouth during administration. Chickens in fair condition will tolerate a dosage as high as 4 cc for each 2.2 pounds of body weight, but doses of this size will result frequently in transient diarrhea and, in the case of laying birds, decrease in egg production for a few days. Such dosage, however, is likely to be toxic for emaciated or very weak chickens.

Prevention.—Poultry should not have access to streams, ditches, or ponds where they can procure tadpoles. Medication should be considered as a preventive as well as a therapeutic measure since, if thoroughly conducted, it prevents the further spread of the infection.

TAPEWORMS

The effects of tapeworm infections in poultry are so insidious that their presence is often entirely overlooked. These worms, inhabitants of the intestines, when full-grown, range from almost microscopic size to 10 inches in length, according to the species. They have a scolex, or "head," by which they attach themselves to the intestinal walls. Attached to the scolex, by a "neck" in most cases, is a series, variable in number, of flattened "segments," or proglottides. They absorb their food directly through the body wall. The posterior segments break off and appear in the feces as glistening white pearly objects which, in freshly voided droppings, can move about slowly by expanding and contracting. These segments contain eggs which are harmless to chickens but when eaten by some other animal which can serve as an intermediate host (see table 1) develop into larval tapeworms. These larval forms remain in the intermediate host until it is eaten by a chicken. Then the larvae attach themselves to the lining of the intestines of the chicken and develop into mature worms (fig. 53).

Amoebotaenia sphenoides is a very small tapeworm. It has from 15 to 30 narrow segments and is found throughout the duodenum. It is relatively uncommon and is seldom of economic importance. Its intermediate host is the earthworm. Although this tapeworm is practically harmless in otherwise normal and healthy poultry, it may aggravate the symptoms produced by other factors which affect the health of the birds.

Choanotaenia infundibulum (fig. 54, *B*) is the most common tapeworm in California. Its specific name relates to the shape of the mature segments, which are funnel-shaped, the posterior ends being wide and invaginated for the reception of the following segment. The larvae of house flies (*Musca domestica*) and the dung beetle (*Geotrupes sylva-*

ticus) serve as intermediate hosts. In house flies, the infection continues through the larval and pupal stages and persists in the adult. *Choanotaenia infundibulum* is not, under normal conditions, a particularly serious tapeworm. The symptoms it produces are similar to those caused by *Davainea proglottina* but are much less severe. This parasite, like the other larger tapeworms, may in heavy infections practically occlude the intestine with the consequent serious disturbance of the normal movement of the intestinal contents.

TABLE 1

Poultry Tapeworms in the United States

Tapeworm	Primary hosts other than chickens	Length when mature segments are formed	Intermediate host
		inches	
Amoebotaenia sphenoides (rare)	None	1/8 – 1/4	Earthworms
Choanotaenia infundibulum (uncommon)	Turkey, Hungarian partridge	3/4 – 10	House fly, dung beetle
Davainea proglottina	Turkey	1/16 – 1/8	Slugs
Hymenolepis cantaniana (rare)	Turkey, peacock, pheasant	1/16 – 1/9	?
H. carioca	Quail, turkey	1 – 3	Dung beetle, stable fly
Raillietina cesticillus (common)	Pheasant, turkey, guinea fowl	3/8 – 5	Ground beetle, dung beetle
R. echinobothrida (cause of nodular taeniasis)	Turkey, pigeon	2 – 10	Ants, land snails, slugs (?)
R. tetragona	Turkey, guinea fowl, quail	1/2 – 10	House fly, ants

Davainea proglottina is one of the smaller tapeworms of poultry, being less than 1/8 inch long and consisting of only 4 or 5 segments, and is considered by some authorities to be the most harmful of the poultry tapeworms. It is found exclusively in the second half of the fore part of the small intestine, deeply imbedded in the mucosa. Its intermediate hosts are slugs. Young birds are more susceptible to this parasite than are older ones. Infected birds lose their appetites, become droopy, are usually thirsty and show anemia and emaciation. Egg production decreases or is even entirely suspended. Diarrhea may occur and the feces may be discolored with blood. Often there are signs of extreme weakness, especially in the legs. Light infections merely result in unthriftiness of the birds, but heavy infections may be fatal.

Hymenolepis carioca, commonly known as the "thread tapeworm of poultry," is about 3 inches long when fully grown, but is threadlike in thickness. Its scolex (fig. 54, *A*) is particularly difficult to obtain at autopsies because the worm breaks readily, and this portion is lost. The intermediate hosts are dung beetles and stable flies (*Stomoxys calcitrans*). This parasite produces symptoms similar to those produced

by *Raillietina cesticillus* and in heavy infections may cause marked symptoms.

Raillietina cesticillus is the most common member of this genus in California. Its scolex (fig. 54, *E*) is flat and inconspicuous and dominated by a protuberance covered with tiny hooks. The suckers are inconspicuous and without spines. The mature worm is from ⅜ to 5 inches long. The young worms are usually attached to the posterior portion of the fore part of the small intestine, but the older worms are more apt to be found in the anterior part of the small intestine. Dung beetles and

Fig. 53.—Inside of a hen's intestine, showing attached tapeworms, two times natural size. At post-mortem examination, the worms appear much longer and more transparent, but they contract and become whiter at death. (From Ext. Cir. 8.)

ground beetles act as intermediate hosts of this tapeworm. The symptoms are like those produced by *R. tetragona* but usually somewhat less severe.

Raillietina echinobothrida (fig. 54, *D*), sometimes known as the "spiny-suckered tapeworm," is one of the most serious tapeworms of poultry. This is one of the larger species of tapeworms found in chickens; it attains a length up to 10 inches and is found in the lower portion of the small intestine. It utilizes ants, land snails, and possibly slugs as intermediate hosts. Infections with this tapeworm give rise to nodules varying from the size of a pinpoint to that of a pea, on the outside of the intestines; this condition is known as "nodular taeniasis." Nodules are often bunched, several coalescing to form what appears to be a single large one. When numerous, they interfere with the nutrition of the bird, and progressive emaciation results. This tapeworm ranks next in seriousness to *Davainea proglottina.*

The nodules produced by this tapeworm so closely resemble those caused by tuberculosis (p. 76) that the former can be distinguished only if the parasites are found protruding from a nodule into the interior of the intestines. Unfortunately, however, after causing a nodule, the worm may change its position, so that at autopsy none will be found attached

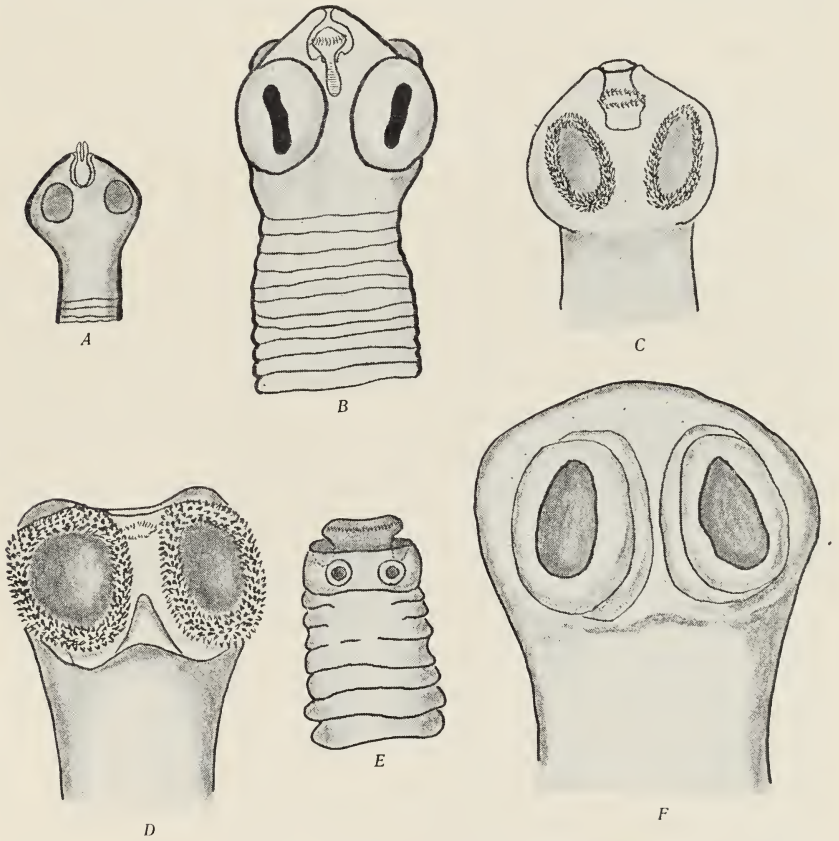

Fig. 54.—Scolices, or "heads," of the common poultry tapeworms greatly enlarged but drawn to the same scale: *A, Hymenolepis carioca; B, Choanotaenia infundibulum; C, Raillietina tetragona; D, R. echinobothrida; E, R. cesticillus; F, Metroliasthes lucida.* (From Ext. Cir. 8.)

to a nodule. Therefore, when nodules on the intestines are found at autopsy of a chicken on the farm, specimens should be submitted to a laboratory (see footnote 14, p. 24) where a definite diagnosis can be obtained.

Raillietina tetragona (fig. 54, *C*) is closely related to the preceding species and, like it, has thorny suckers; it is about the same size and inhabits the same portion of the intestine. It utilizes house flies and ants as intermediate hosts. This parasite produces in poultry symptoms simi-

lar to those described for *Davainea proglottina* (p. 137) and is nearly as serious; it does not produce nodules.

Diagnosis.—At post-mortem examination the lining of the small intestine is seen to be thickened and may be bleeding. The presence of the parasite is easily overlooked because it is partially embedded in the tissue and is frequently too small to be readily recognized. If scrapings from the infected intestinal lining are placed in water in a flat dish, however, the worms can be detected easily. Diagnosis can also be made by finding the eggs in the droppings, under microscopic examination; especially in droppings samples passed in the early afternoon.

Prevention.—In order to prevent infections with tapeworms, chickens must be kept from eating the intermediate hosts (see table 1), preferably by exterminating the latter.

When slugs are involved as intermediate hosts, they may be controlled in a given area by the use of a poison bait. One-quarter ounce of metaldehyde ground up and thoroughly mixed with 8 ounces of bran makes a satisfactory bait. After the metaldehyde and bran are mixed, sufficient water is added to make the bait moist. The bait is put out toward evening in small piles of approximately 1 teaspoon each; one pile to about each 2 square feet of soil surface. Care must be taken in dry areas to keep the baits moist. The metaldehyde baits which also contain calcium arsenate are more efficient than those without the arsenical. A very satisfactory bait formula is the following: 2 tablespoons blackstrap molasses, 1 ounce calcium arsenate, ½ ounce metaldehyde, 1 pound bran, 1 pint water. In making this latter bait, the calcium arsenate and metaldehyde are first thoroughly mixed with the bran and then the molasses, dissolved in the water, is added. Chickens possess a marked tolerance to arsenicals and are not poisoned by metaldehyde, but despite this, baits placed in chicken yards or other places to which chickens have access should be picked up each morning before the birds can feed upon them.

There is no known practical method of chemical control of earthworms, but their abundance may be reduced to a negligible quantity by keeping the runs dry and well drained, avoiding the spilling and accumulation of water from drinking fountains, and preventing the accumulation of manure.

House flies, which serve as intermediate hosts for some species of tapeworms (table 1), may be controlled as described on pages 130–132.

The prompt and effective disposal of manure, required in house-fly control, will also eliminate dung beetles, which harbor the immature stages of certain tapeworms, since they will thereby be deprived of their source of food supply.

When ants serve as intermediate hosts for tapeworms, they may be

controlled by the proper employment of a state or federally approved ant poison. Precautions must be taken, however, when materials poisonous to chickens are used that the birds do not have access to the poison.

Ground beetles are difficult to control, and the control method varies with the habits of the different species. When these insects are involved in tapeworm infections, specimens should be submitted to an entomologist for identification and advice as to appropriate control measures.

The control measures for stable flies are directed toward their breeding places. Moisture is necessary for the development of the larvae; dry material is not suitable for their needs. The more important breeding places of these flies can be controlled by promptly removing moist feed wastes from poultry yards and near-by feed lots and stables, and scattering them to hasten drying or destroying them. Lawn cuttings, weed piles, vegetables, vegetation washed up on lake shores, and other plant materials and wastes must not be permitted to accumulate in piles long enough to ferment and decay. Loosely piled strawstacks, if allowed to become wet, are important breeding places of the stable fly. Hence, straw for feeding and breeding purposes should be baled and stored under cover or the stacks should be rounded up so as to make the top largely rainproof and the sides nearly vertical.

Treatments.—There is no drug known to be satisfactory for the treatment of poultry-tapeworm infections. Earlier experiments indicated that kamala was an effective drug for this purpose, but subsequent investigations have shown that the scolices ("heads") are not removed and that new segments are promptly regenerated. Since the damage to the host is caused almost entirely by the scolices, it is futile to administer kamala to infected birds. Colloidal iodine (see p. 17) has been recommended for the treatment of tapeworm infections in chickens, but investigations have shown that this drug removes comparatively few scolices. No treatment for poultry-tapeworm infections can therefore be recommended until one that will reach and kill the scolices has been discovered.

GENERAL CHARACTERISTICS OF ROUNDWORMS

The name "roundworm" is commonly applied to the slender wirelike worm, *Ascaridia galli,* which is so frequently found in the intestines of poultry. This term, however, actually applies to a very large group of worms, a number of species of which are parasitic in domestic birds. These worms are unsegmented, usually cylindrical, and elongate in shape. With but few exceptions the sexes are separate. An intermediate host may or may not be required.

THE LARGE ROUNDWORM OF POULTRY

The large roundworm of poultry, *Ascaridia galli,* as mentioned above, is a common parasite of the intestines. It is slender and wirelike and attains a length of from 1 to 4⅘ inches when fully grown. The mature worms in the intestine deposit eggs which pass out of the body of the birds in the droppings. After about 10 days' exposure to the air, a young worm develops in the egg, which, if eaten by a bird, promptly hatches in the intestine. After about 10 days the resultant worm invades the intestinal mucosa. Seven days later it returns to the intestinal tract. Only very exceptionally, and then accidentally, does a roundworm migrate through the lungs. The worm matures in from 5 to 8 weeks after the egg has been swallowed.

The eggs escaping from the birds are very resistant and will remain infective in the ground in shaded places for over 3 months. Under experimental conditions, they are rapidly killed by dry, hot weather, even when buried 6 inches deep in soil exposed to sunlight, but this does not seem to apply under average farm conditions.

It is believed that the worms probably produce a toxic substance, or substances.

Symptoms and Diagnosis.—The symptoms of large-roundworm infection in poultry may become apparent within the first week of infection. The feathers are ruffled, the wings droop, the birds assume a hunched-up drowsy attitude and become emaciated and anemic. The appetite is at first diminished, but later the birds may become voracious. They will remain stunted, however, despite the large appetite. The young parasites may produce marked lesions when they penetrate into the intestinal lining. These lesions cause hemorrhage and inflammation and the birds become anemic, with marked paleness of the comb and shanks, and suffer from diarrhea. The birds are unthrifty, generally weak and sometimes paralyzed, and egg production is decreased. In heavy infections, the worms may cause intestinal obstructions. Young birds are more susceptible to this parasite than are adults or those which have had a previous infection. Proper bone development is prevented in the young birds. Vitamin-A and -B deficiencies make chickens more susceptible to this worm. Diagnosis is made by finding eggs in the feces by microscopic examinations or by finding the parasites in the intestine at autopsy (fig. 55).

Source of Infection.—Large-roundworm infections are acquired by poultry through consumption of eggs in droppings or soil or by feed and water contaminated by egg-bearing droppings. Earthworms in contaminated soil may mechanically carry eggs and infect chickens which

eat them. They do not, however, serve as intermediate hosts as they do for tapeworms.

Treatment.—Nicotine, although very poisonous for chickens, is a highly effective drug in the treatment of large-roundworm infections. When it is combined with certain protective colloids, an effective dose of the drug can be safely given. Such preparations are readily available from commercial sources in either pill or tablet form for individual treatment and in a powdered or granular form to be mixed with the

Fig. 55.—Section of intestine showing roundworms, *Ascaridia galli*, protruding from the cut ends. (From Ext. Cir. 8.)

mash for flock treatment. Individual treatment is preferable, because when mixed with the mash the nicotine may volatilize and be lost and the more badly infected birds of a flock are likely to have poor appetites and therefore get a smaller dose of the drug than the more vigorous birds which need the treatment the least. Such uneveness in dosage from flock treatment can be partially overcome by separating the weak from the vigorous birds before the medicated mash is given.

A nicotine preparation of this type consists of a mixture of 6.2 cc of Black Leaf 40 with 16 grams of a special fuller's earth known as Lloyd's Alkaloidal Reagent. The powder is packed into no. 2 gelatin capsules in sufficient quantities to make the filled capsule weigh from 350 to 400 milligrams. Lloyd's Alkaloidal Reagent has the property of protecting the treated birds from the toxic action of the nicotine and yet liberating sufficient amounts of nicotine in the intestines to eliminate

the worms. Most other fuller's earths lack this property and, although less costly, cannot be substituted. These capsules are available from commercial sources, usually under the name of "University Capsules." The dose is 1 capsule to a bird, placed well along the throat and, with the thumb and forefinger on the outside, slid down into the crop. The dosage is one quarter to one half less for chickens under 8 weeks of age. These capsules may be poisonous to chickens that are emaciated or that have been shut in the houses overnight without feed or water and therefore have empty crops.

Another, though little used, method of giving nicotine is by feeding a mash containing 2 per cent tobacco dust continuously for 3 or 4 weeks. The tobacco dust should be a brand marketed in tightly sealed packages and have a guaranteed nicotine content of 1.5 to 2.0 per cent. Since the nicotine may be rapidly lost from tobacco dust on exposure to the air, only a small quantity of the medicated mash should be mixed at a time.

Another effective treatment for large-roundworm infections is carbon tetrachloride. The drug may be given in capsules, employing the same technique as for nicotine capsules, the first thing in the morning after starving overnight. Care must be exercised not to break the capsule in the mouth since death may quickly result in such an event. The recommended dosage of carbon tetrachloride is as follows: 2 cc for birds 6 months and older; 1½ cc for birds 4 to 6 months; and ½ to ¾ cc for birds under 2 months. For chickens 2 months old or younger, it is perhaps better to administer tetrachlorethylene in the same dosage as for carbon tetrachloride. Instead of administering these drugs in gelatin capsules, they may be introduced directly into the crop by means of a syringe and a piece of soft rubber tubing. A graduated glass-barreled 5- or 10-cc syringe or a hard-rubber rectal syringe and a piece of no. 5 gauge rubber tubing about 6 inches long are required. The bird's mouth is opened and the tubing is inserted down the throat into the crop, taking care to prevent the tube from entering the windpipe. The syringe is then filled with the drug and attached to the tubing and the recommended dose is injected. The syringe is detached and the tube is withdrawn very slowly to permit all of the liquid to run out. Carbon tetrachloride or tetrachlorethylene should not be administered to birds that have been fed a diet deficient in calcium.

It is felt that flock treatment is unwise because the dosage cannot be controlled as it can be when birds are treated individually. Despite this, however, flock treatment is not uncommon. Flock treatments that can be used are 1 teaspoon of oil of chenopodium, thoroughly mixed with a moist mash, for each lot of 12 birds, tobacco dust, or the commercial nicotine preparations.

Prevention.—The procedure described for the prevention of coccidiosis (p. 92) applies perfectly to large-roundworm control. In the prevention of this infection, it is particularly important that all wet spots in the yards and ranges be eliminated. Marked results have followed the installation of gravel pits covered with narrow cleats under all drinking fountains. Moist soil serves as an incubator for the worm eggs deposited in the droppings of infected birds. The rotation of runs has been found to be of considerable value in preventing infection with this parasite. If young birds can be kept free of the worms for the first 3 months of their lives by confining them in houses or on clean ground, they are able to resist infection when placed on infective premises, because of a developed "age resistance," to such an extent as to prevent the greatest injury. This resistance, however, is dependent upon their having received a nutritious diet, rich in vitamins.

As soon as the parasites are eliminated by treatment of the birds, the droppings boards and pens should be cleaned up to prevent reinfection from worm eggs already on the premises. (The eating of the roundworms by chickens immediately after elimination is not dangerous because the eggs contained in the female worms are not infective until they have been exposed to the air for about 10 days.) It is best to confine the chickens to houses with sun porches. If this is impossible, it is advisable to remove surface dirt and droppings from the entire yard in the case of small yards, or, in the case of large yards, from the areas where the birds congregate immediately after treatment, such as around water supplies and close around the houses. Plowing such places is profitable. No known chemical spray is effective in killing roundworm eggs.

THE CECUM WORM

The cecum worm, *Heterakis gallinae,* is a grayish-white worm from ⅓ to ½ inch long, found in the ceca, or "blind gut," of the intestines. Because of their small size, they are easily overlooked or may be mistaken for the young of the large roundworm. The eggs (fig. 56) are voided in the feces, develop in the open, and reach the infective stage in 7 to 12 days or longer. These eggs are very resistant. When a bird swallows an infective egg, it hatches in the intestine after from 1 to 2 hours.

Symptoms and Diagnosis.—The cecum worm is of little importance in older birds, in which heavy infections are rarely seen, but in younger ones they may interfere with the normal functioning of the ceca and in baby chicks may be very harmful and cause death as early as the tenth day. The particular importance of this worm, however, is that it is involved in the transmission of blackhead of turkeys.

Sources of Infection.—Infection with the cecum worm is acquired in the same manner as described for the large roundworm (p. 142).

Treatment.—Because of their sheltered position in the ceca, these parasites are very hard to remove. Tobacco dust will remove about 80 per cent of the cecum worms present when it is fed as 2 per cent of the mash over a period of a month. The tobacco dust should be a brand marketed in tightly sealed packages and have a guaranteed nicotine content of 1.5 to 2.0 per cent nicotine. Good results have been reported with phenothiazine given at the rate of 0.5 gram per bird.

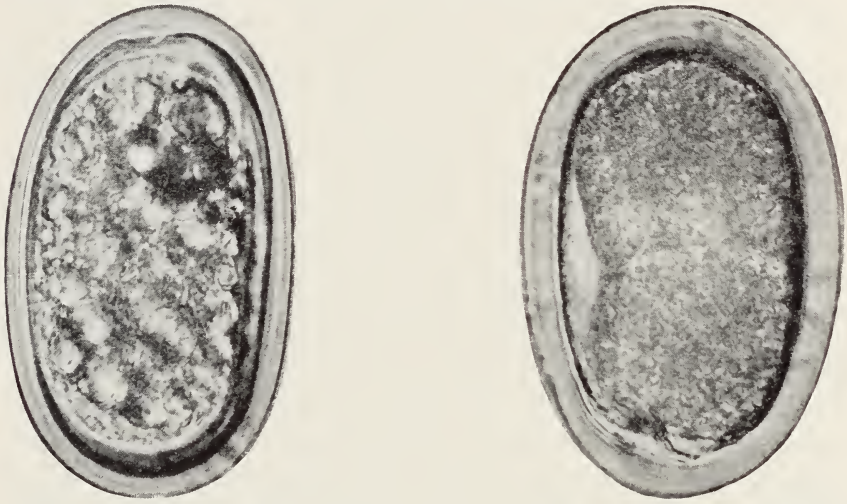

Fig. 56.—Highly magnified eggs of cecum worm (left) and intestinal roundworm (right), which pass out in the droppings. (From Ext. Cir. 8.)

Rectal injections of oil of chenopodium in a bland oil such as cottonseed oil will remove about 90 per cent of the parasites. One tenth of a cc of oil of chenopodium in 5 cc of cottonseed oil is administered to a bird weighing 1½ pounds. Probably double this dose would be indicated for a bird weighing 3 pounds or more. The liquid should be thoroughly mixed and administered with a hard-rubber rectal syringe. The tip of the syringe should be covered with oil and gently inserted into the rectum, and the fluid should be injected slowly.

Prevention.—The preventive measures to be taken against cecum-worm infections are the same as those described for the prevention of large-roundworm infections (p. 145).

THE GIZZARD WORM

The gizzard worm, *Acuaria* (*Cheilospirura*) *hamulosa,* is a fairly slender reddish worm, from ½ to ¾ inch long, frequently found coiled in the muscular coat of the gizzard. The eggs of this parasite are passed

out in the feces. If they are eaten by a grasshopper, they hatch, and the larvae pass through three developmental stages in this intermediate host. The infective larvae gain access to poultry when the infected grasshoppers are eaten. The development in the grasshopper requires 22 days, and 76 days are required for the worm to come to maturity after being ingested by the chicken. Certain species of beetles and sandhoppers may also be involved as intermediate hosts of this parasite.

Symptoms and Diagnosis.—Mild or even moderately heavy infections do not appear to be harmful. Very severe infections, however, may interfere with the food-grinding function of the gizzard and thus cause emaciation and weakness. The presence of gizzard worms in an emaciated bird does not necessarily indicate that the emaciation is due to the effects of the parasites, and further search should therefore be made for other causes.

Diagnosis may be made at autopsy by finding the worms beneath the lining of the gizzard. They are most frequently found near the entrance of the stomach, and their presence is denoted by blotchy discoloration of the lining of the gizzard. Diagnosis may also be made by detecting eggs in the feces.

Treatment.—Because these parasites lie concealed in the muscle tissue of the gizzard, they are protected from the action of drugs and no effective treatment is known.

Prevention.—Preventive measures consist in disposing of the droppings in such a way that grasshoppers will not feed upon them, not allowing the birds to range where grasshoppers are plentiful, and taking steps to reduce the number of grasshoppers.

Farm practices which are helpful in controlling grasshoppers are (1) cultivation of infested fields and adjacent ditch banks in the late fall and early winter to destroy the eggs of the insects; (2) rotation of crops where alfalfa has been raised continuously for five or more years to destroy eggs and provide conditions less favorable for grasshopper increase; and (3) baling alfalfa hay at night so that grasshoppers roosting in the haycocks will be included in the bale. The latter practice may decrease the grasshopper population as much as 75 per cent.

Poison baits are very effective in controlling grasshoppers. Any of the following poisons have been proved to be very effective when mixed with 100 pounds of bran and 12 to 16 gallons of water: 2 pounds paris green (50 per cent As_2O_3) or 3 pounds commercial arsenic (98 per cent As_2O_3) or 2 pounds powdered sodium arsenite (85 per cent As_2O_3). The soluble arsenicals, paris green and sodium arsenite, are dissolved in the water before being mixed with the bran. Commercial arsenic, which is insoluble, is thoroughly mixed with the bran before the water is added. The efficien-

cies of these poisoned baits depend upon thorough mixing and the proper time of application. They should be put out, preferably by broadcasting on large areas and in small closely set piles on small areas, early in the morning when soil temperatures are between 60° and 75° F. Since the maximum feeding period of the insects is when the temperature is between 70° and 85°, it is not wise to put the baits out when the temperature is above 85°. Although chickens have a marked tolerance to arsenic, they should be prevented from feeding upon these poison baits.

The sandhopper is not an insect but is related to the shrimp. It is small, pale, and shrimplike in appearance and breeds in areas which are shaded and moist. Where sandhoppers serve as the intermediate host for gizzard worms, chickens should be kept away from such areas to prevent infection with this parasite.

HAIRWORMS, OR CAPILLARIA WORMS

Several different species of worms belonging to the genus *Capillaria*, called Capillaria worms, or hairworms, have been reported from poultry. These parasites are all extremely slender and are usually from ½ to ¾ inch long, though the largest species may attain a length of 3 inches. Even the larger ones, however, are difficult to detect at post-mortem examination because they are slender and imbedded in the mucosa. *Capillaria annulata* invades the crop and esophagus; *C. retusa* and *C. dubia* the ceca; *C. longicollis* the duodenum; and *C. columbae*, *C. collaris*, *C. gallinae*, and *C. caudinflata* the small intestines. The eggs of all of these species are oval with transparent pluglike structures at each end. With all but *C. annulata* the eggs, as soon as they have reached a certain degree of development after elimination with the droppings, are apparently directly infective to other susceptible birds without the intervention of an intermediate host. Recent investigations, however, indicate that earthworms serve as the intermediate host of *C. annulata*.

Symptoms and Diagnosis.—Hairworm infections in poultry may seriously affect the health of the birds. Heavy infections produce emaciation, weakness, and death. Affected birds become depressed, the feathers are ruffled, and the wings droop. A severe inflammation may be produced and the droppings may contain pinkish shreds of mucus. Diagnosis is usually made at autopsy by finding the parasites, though this may be difficult unless they are very numerous. Detection of the parasites at post-mortem examination is facilitated by immersing the open section of the affected portion in water and carefully tearing the lining. Diagnosis may also be made by observing the eggs in the droppings.

Source of Infection.—Capillaria-worm infections are acquired by ingesting infective eggs on contaminated soil, as described for the large

roundworm (p. 142). In the case of *Capillaria annulata,* infection may possibly be acquired through the ingestion of earthworms.

Treatment.—There is no treatment known that is entirely satisfactory for hairworm infections in poultry. Carbon tetrachloride given, as described on page 144, in doses of 1 cc, and repeated after about 7 days, is effective in removing some species of *Capillaria* worms.

Prevention.—The preventive measures to be taken against hairworm infections are the same as those previously described for large-roundworm infections (p. 145). In the case of *Capillaria annulata* infections, it may be necessary to avoid contact of the birds with earthworms by the methods described on page 140.

THE GAPEWORM

The gapeworm, *Syngamus trachea,* which is extremely rare in California, is a small reddish worm found in the windpipe. The female, which is ½ to 1 inch long, is attached to the lining of the windpipe. The male, which is seldom more than ¼ inch long, is attached to the female in copulation in such a way as to form a Y. The male is also attached to the tracheal wall, where it usually forms a nodule. The eggs deposited by the female worms pass up the windpipe with mucus, are swallowed, and expelled from the body with the droppings. After from a week to a month, according to the temperature, in the open air, a fully developed embryo capable of infecting a healthy bird is formed. This may be eaten, while still confined within the egg, by the bird, or it may hatch and be eaten as a free-living larva. Earthworms feeding on poultry manure may eat either infective gapeworm eggs or larvae, in which case they may live for some time in the alimentary canal of the earthworm and thereby be protected from drying or other unfavorable environmental conditions. The young gapeworm, when eaten by the chicken as a developed, infective egg or a free-living larva from contaminated soil, feed, or water, or in the body of an earthworm eaten by a fowl, penetrates the walls of the alimentary canal and reaches the lungs through some as yet unknown route. Here it undergoes a period of development, after which it migrates up the bronchi to the trachea. The trachea is reached as early as the seventh day after infection and eggs appear in the feces from the seventeenth to twentieth day. Some investigators claim that passage through earthworms renders the larvae more highly infective. The parasites suck blood while attached to the wall of the trachea.

Symptoms and Diagnosis.—Mature birds rarely become infected with gapeworms, and when they do the parasites rarely remain in the trachea for more than a month. Consequently the mature chickens in an infected

flock show only mild symptoms or none at all. Young chickens, however, may be severely affected. The trachea becomes partially clogged by the parasites and mucus, with resultant gasping for air, or gaping, and spasmodic coughing. The affected birds refuse food and water and become emaciated and weak. Some may die from suffocation.

Treatment.—The mechanical removal of gapeworms from the windpipes of poultry with clipped feathers, horsehair loops, and patented wire extractors is rather widely practiced but is a slow, tedious, unreliable, and somewhat dangerous practice. Of various drugs which have been recommended for the treatment of this infection, barium antimonyl tartrate is by far the most satisfactory. The birds to be treated are placed in a relatively small closed box or other container that is deep enough to have a space of at least 6 inches above the heads of the birds when they are standing erect. The container should be small enough to be tilted from side to side or be equipped with a small electric fan. One ounce of the finely powdered chemical for each 8 cubic feet of space within the container is required for each group of birds to be treated. One third of the total amount of the barium antimonyl tartrate to be used is put into the box through an opening in the top by means of a dust gun. The container is then tilted from side to side or the electric fan is started to keep the powder dispersed in the air and to cause the chickens to breath more deeply and frequently. Five minutes later a second third of the powder is introduced and the container is tilted or the electric fan is started again. In another 5 or 10 minutes, the procedure is repeated with the third portion of the powder. The birds are released 5 to 10 minutes after the last portion of the powder is blown into the box.

The barium antimonyl tartrate should be prepared by a druggist or chemist in the following way: A barium chloride solution is made by dissolving 244.3 grams of crystallized barium chloride in 800 cc of distilled water at a temperature of 70° to 75° F. This solution is then mixed slowly with a warm solution of potassium antimonyl tartrate, made by dissolving 667.8 grams of the chemical in 1,700 cc of distilled water. The mixture of the two solutions is then stirred vigorously with a glass rod. A precipitate is formed and is collected by filtering immediately through paper with the aid of suction. The precipitate thus collected is washed with cold distilled water and then with alcohol to facilitate drying. The washed precipitate is dried at room temperature, since higher temperatures while the chemical is wet may cause decomposition. The yield of barium antimonyl tartrate is approximately 1¾ pounds. If properly prepared, the finished product will pass through a 100-mesh sieve; otherwise it must be ground to that degree of fineness.

Prevention.—The most effective preventive measure is to raise the young birds apart from the older ones and on land not previously occupied by gapeworm-infected birds. Land on which infected birds have not run for at least two years may be used without immediate danger of losses from gapeworm infections. Sandy, well-drained soil is highly desirable, since soil of that type is not favorable for development of gapeworm eggs and larvae and is less likely to be infested with earthworms. All moist localities and situations such as wet grass should be avoided to prevent feeding upon earthworms. Confinement of the birds to houses with sun porches and the regular removal of droppings and contaminated litter at least at weekly intervals would afford ideal protection against this parasite.

Turkeys of any age are susceptible to gapeworm infection but they show no symptoms and therefore are dangerous carriers of the disease. Consequently turkeys should not be kept with chickens nor should chickens be placed on land that has been occupied by turkeys.

Infected birds that cannot be treated should be killed and destroyed by burning.

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